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# HANDBOOK

ON

# CONTAGIOUS AND INFECTIOUS DISEASES IN ANIMALS

ISSUED BY THE

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## PREFACE.

This Hand book has been compiled in the Veterinary Section of the Quartermaster General's Branch, General Headquarters, India, and is intended as a guide to all concerned in the suppression of contagious diseases in animals.

It was first published in 1910, and revised in 1928, 1936, 1942 and 1944.

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# HANDBOOK

## ON

# CONTAGIOUS AND INFECTIOUS DISEASES IN ANIMALS

## PART I.

### A FEW FACTS REGARDING MICRO-ORGANISMS.

Micro-organisms, popularly referred to as microbes, germs, bacteria etc., cause contagious diseases. The symptoms seen in these diseases are the results of the invasion of the animal body by these parasites.

It follows therefore that if outbreaks of contagious disease are to be dealt with and suppressed, something must be known of the life history of the micro-organisms which cause them.

They are generally classified as the lowest order in the plant world but some, *e.g.*, the protozoa belong to the animal kingdom.

**Structure of micro-organisms.**—Bacteria appear under the microscope as pale translucent bodies; they are one-celled organisms composed of protoplasm surrounded by a membrane or skin which, in some, swells up to form a jelly-like casing.

**Types of organisms.**—The organisms vary very much in shape and size. Some are globular or spherical in shape, and are generally known as cocci; many, on the other hand, are rod-like bodies, hence are termed bacilli; whilst others, having a spiral or corkscrew shape, are known as spirilla. Long unbranched filaments are called leptothrix, while others which branch are known by the name cladothrix. All these various shaped organisms are loosely spoken of as bacteria.

Other classes of micro-organisms, are the moulds and the saccharomycetes or yeasts. Moulds consist of slender threads which give rise to the woolly patches often seen on various articles of food; the saccharomycetes or yeasts are ovoid or sausage-shaped cells which are much larger than the bacteria proper.

The protozoa referred to above cause trypanosomiasis in man and animals, the conditions known as sleeping sickness in man and "surra" "nagana" etc., in animals being closely allied.

**Size of organisms.**—Most of the bacteria are on an average from $\frac{1}{25,000}$ of an inch long to about five times that length. The best impression of the size of bacteria is obtained when it is stated that a $\frac{1}{25}$ inch immersion lens (microscope) gives a magnification of nearly 2,200 diameters,

and that under this power the bacteria appear to be about the size of ordinary print. If we could view the average human being under such circumstances he would appear to be about four miles in height. The standard of measurement is the micron, represented by the Greek letter $\mu$. One (micron is equal to about $\frac{1}{25,000}$ of an English inch. Some micro-organisms are so small that they are beyond the range of vision of our most powerful microscopes, and have not yet been discovered. Included in this category of "ultravisible viruses" are the causal agents of Rabies, Variola, Cow-pox, Sheep-pox, Foot and Mouth disease, Rinderpest, South-African horse sickness, Canine distemper and probably Swine fever. The causal agent in the majority of these cases passes through the pores of the finest porcelain filters.

**Movement of Micro-organisms.**—Many of the bacteria are motile, especially bacilli and spirilla. The movement, in most, is induced by one or more hair-like processes termed flagella attached to the ends of or all over, the organism. Other micro-organisms particularly cocci, are quite motionless.

**Methods of reproduction.**—This takes place by "fission" or by "spore" formation. Fission is transverse division into two parts, the organism first elongating and then becoming narrower and narrower in the middle until the two halves become free. If the organisms are cocci and divide irregularly they may form clusters resembling bunches of grapes and are then known as staphylococci; if they remain connected in the form of chains like strings of beads they are known as streptococci. If the division of cocci takes place regularly and in one plane, diplococci are formed; if division takes place in two directions, tetra-cocci or tablet-cocci are formed; and if the division is in three directions and in two planes, sarcinæ or packet-cocci are the result.

Spore formation may take place in two ways, namely, by "endogenous spores" and "arthrospores". In the former, a round or ovoid highly refractile body developes in the protoplasm of the micro-organism. This is the spore. The enclosing membrane of the micro-organism breaks away and the spore becomes free. In the latter (Arthrospores) the entire cell or organism owing to lack of favourable conditions of growth, becomes converted into a spore.

It should be noted that the anthrax bacillus forms spores only in the presence of oxygen. These spores are much more resistant to disinfectants than the vegetative form of the bacillus, so care should be taken to avoid spilling blood in cases of anthrax.

**Growth of Bacteria.**—Bacteria multiply with incredible rapidity under conditions favourable to their growth and development, but fortunately various checks such as lack of suitable nutriment and unfavourable physical conditions prevent unmanageable multiplication.

All bacteria require water, salts, carbon and nitrogen for their growth. As they do not contain chlorophyll they are unable to avail themselves of the carbon existing in the air as carbonic acid gas, but are dependent for their nourishment on organic matter, obtaining carbon from

complex compounds of carbon such as the sugars, and nitrogen from nitrogenous compounds in the shape of the albuminoids. Some, however, may obtain their nitrogen from inorganic materials such as compounds of ammonia and nitrates.

Some bacteria require oxygen for their growth and are known as **aerobes;** others can only live in the absence of oxygen, and are termed anærobes; while still a third group can live either in the presence or absence of oxygen.

Heat is necessary for the development of bacteria. It has been found that disease-producing or "pathogenic" bacteria thrive best at the temperature of the animal body.

Light is, as a rule, unfavourable to the growth of bacteria, and direct sunlight frequently kills them. Chemical agents cause their destruction, and this is the basis of disinfection.

According to their conditions of life, bacteria may be broadly divided into two classes. When their nourishment is drawn from some living body or "host", they are known as "parasites". They are further termed "obligatory" parasites if they can only live on this host. If the bacteria draw their nourishment from dead organic matter they are called "Saprophytes". This phase in the life history of micro-organisms has a great bearing on the successful and permanent extinction of contagious disease and should be carefully considered. Pathogenic organisms which depend entirely on a host for their existence can be easily dealt with, but not so those which can continue their existence in saprophytic conditions.

## Infection—how bacteria produce disease.

Pathogenic organisms may exert their pernicious power in several ways. They may be injurious on account of their abstracting nourishment from the blood and tissues, or by mechanically blocking up the minute capillaries and blood vessels by their excessive multiplication. But their main poisonous action is due to the secretion or excretion of chemical products. These products are termed "toxins", and it is by their circulation and absorption within the body that disturbances of the animal system characterizing disease are caused.

Infection may result through—

(*a*) Wounds, *e.g.*, Rabies, Tetanus, Epizootic Lymphangitis.

(*b*) Inhalation, *e.g.*, Influenza.

(*c*) Ingestion *e.g.*, Rinderpest, Foot and Mouth disease, Glanders.

(*d*) Bites of insects, *e.g.*, Surra, Biliary Fever and other Piroplasmoses.

(*e*) Coitus, *e.g.*, Dourine.

## Immunity and how obtained.

Immunity is the ability of an individual or species to resist infection. The resistance offered may either be against the organisms or against their toxins. Immunity is **natural** or may be **acquired.** The lower animals are naturally immune to the majority of contagious human diseases and *vice versa*.

When an organism is capable of producing specific disease in an animal, that animal is said to be "*susceptible*" to that disease. Susceptibility varies in degree even in the same kind of animal, the important factors regulating such being—

(*a*) The age of the animal, young animals being often more susceptible than those fully grown.

(*b*) The condition of the animal's health, robust animals resisting disease which debilitated animals may readily contract. In this connection it is important to remember that when contagious or infectious disease prevails, whether amongst human beings or animals, causes of exhaustion, debility, or other conditions calculated to lower the resistance of the system against attack by the microbial cause of the disease should be avoided.

This is specially important in equine Influenza.

(*c*) The manner in which infection is presented, whether aerially, by ingestion, or by inoculation, the two latter being most frequent in the lower animals.

"**Acquired**" immunity may be "**active**" or "**passive**". The former is of long duration; the latter is transient (two to three weeks).

*Active* immunity is conferred by—

(*a*) A previous attack of the disease, *e.g.*, Rinderpest, Variola, Strangles.

(*b*) Inoculation of the pure virus, *e.g.*, pleuropneumonia Contagiosa and Sheep-pox.

(*c*) Inoculation of attenuated (weakened) virus.

The virus may be attenuated by—

(1) Heat, *e.g.*, Anthrax, Quarter-ill.

(2) Drying, *e.g.*, Rabies.

(3) Passing it through other animals, *e.g.*, Swine erysipelas. (Through Rabbits.)

(4) Addition of chemical agents, *e.g.*, Tetanus, a solution of Iodine being used.

(5) Prolonged cultivation on artificial media, *e.g.*, Fowl cholera.

(6) Unknown causes in the bodies of sick or recovered animals, *e.g.*, Rinderpest bile, or the blood of recovered cases of Texas fever.

An attenuated virus is termed a "Vaccine". The use of vaccines for the prevention of disease is year by year becoming more general. The usual procedure is to begin with a very weakened virus and to increase the strength at successive inoculations until the pure virus can be successfully resisted.

(*d*) Sero-virus inoculation, the serum of an immunised animal and virus of the disease being used either simultaneously (*e.g.*, serum on one side of the body and virus on the other) or serum first followed by virus.

"*Passive*" immunity is produced by inoculation of varying quantities of *serum* taken from an animal immunised by any of the above-mentioned methods of conferring active immunity. The immunity conferred by the use of a serum is very transient, and therefore for

prevention of disease it is only of value during an outbreak. Its action is soon established, and we are enabled to tide over a critical time (*e.g.*, Rinderpest).

In outbreaks it is often necessary to repeat serum inoculations (*e.g.*, Rinderpest every ten days). Sera are also of great value as curative agents, and are the basis of the modern system of serum-therapy. Their action may be directed against *toxins* of disease germs (anti-toxic sera) or the germs themselves (anti-microbic sera).

If the serum is obtained by inoculating an animal with one strain only of a particular organism, it is termed a **Monovalent serum;** if it is furnished from several different strains of the same species of microbe it is termed a **polyvalent serum,** *e.g.*, the Pasteurella group of diseases.

## Theory of immunity.

There are various theories to explain the practical phenomenon of immunity. These are:—

(1) **Metchnikoff's Phagocytosis Theory,** which attributes immunity to the action of living cells of the body, *viz.*, phagocytes or leucocytes. The phagocytes act the role of policemen. They are attracted towards invading material by what is termed chemiotaxis, and operate either by incorporating and digesting the micro-organisms, or by giving origin to products which render their toxins inert.

(2) **Ehrlich's lateral Chain Theory,** or the Humoral Theory, which attributes protection to a **Chemical** neutralization through the agency of extra-cellular fluids, notably the blood serum. It is assumed that the normal protoplasm of the body cells is built up of complex organic molecules consisting of—

(*a*) A central stable group.

(*b*) Lateral, less stable, side chains or receptors. Under ordinary circumstances these lateral chains take up molecules of food for nourishment of the cells, but they may also unite with toxin molecules or bacteria causing death of the side chains or the cells themsleves and setting up pathological conditions varying in degree more or less according to dosage. If the dose of toxin is not too great, recovery ensues, and it is legitimately assumed that recovery is attended with reproduction of fresh lateral or side chains. This may be repeated again and again, as in the process of conferring active immunity, until, following the physiological law of Weigert where continued stimulation is followed by over production, so many side chains are formed that they can no longer remain attached to the cell, and they are consequently cast off, excreted as it were, and float free in the blood. These free receptors or side chains are conceived by Ehrlich to be the anti-bodies or antitoxin, and they unite with the toxin before it reaches the cells, thus protecting the latter.

This theory is easily understandable in regard to the neutralisation of toxins of bacteria, but for immunity against bacteria themselves the process is a little more complex. Bacteria must be destroyed. and it is assumed that the antibodies (termed also amboceptor, immune body)

thrown off into the blood, have a sensitising or mordant action on the bacteria whereby a normal constituent of blood plasma termed Alexin is enabled to bring about destruction.

(3) **The Opsonic Theory,** which is a combination of the two foregoing theories. In this theory it is affirmed that the phagocytes are unable in themselves to pick up and destroy micro-organisms, but before phagocytosis can take place, the micro-organisms are acted on or "prepared" by certain constituents of the body fluids termed opsonins ("feast preparers").

Resistance to disease or otherwise is therefore rated in accordance with an opsonic index or value, high in the case of immunity and low in cases when a disease is contracted. During immunisation by sera and vaccines the opsonic index is lowered for the first few days, then increased, remaining at a high level for varying periods of immunity. Care should therefore be taken when these are used not to expose animals to infection during the period when the opsonic index is reduced.

**The Bacteriophage. (Twort-D'Herelle Phenomenon).**—It has been demonstrated that in a human being convalescent from bacillary dysentery, this individual's faeces, when emulsified in broth and filtered through a porcelain filter, and then introduced into a pure culture of the dysentery bacillus in broth and incubated for 24 hours at 37′C., destroyed the dysentery bacilli and the broth is rendered clear.

The smallest drop from this tube containing these dissolved and destroyed bacilli is capable of continuing the phenomenon of dissolution in other pure broth cultures of the bacillus of dysentery.

The filtrate of faeces can be conserved for a considerable time.

The filtrate therefore contains something which has the power of killing these bacilli and to this has been given the name **Bacteriophage.**

**Nature of Bacteriophage.**—D'Herelle considers that Bacteriophage is a living ultra-visible microbe attacking and destroying bacilli.

The Bacteriophage multiplies only at the expense of living and normal bacteria, and may be considered as an internal parasite penetrating the interior of the bacterium, multiplying and secreting ferments and forming a colony of 15-20 individuals. These individuals eventually rupture the bacterium and the young ultra-microbes proceed to attack other bacteria.

**Properties of Bacteriophage.**—It will pass through the pores of a filter and will do so more readily than some of the filterable viruses. It is destroyed by ordinary disinfectants less readily than are bacteria and more readily than spores. Filtrates and cultures can be preserved for six years in a sealed tube. The theory of its living nature is supported by the possibility of demonstrating its power to increase its concentration at the expense of growing bacteria in suitable media.

**Isolation of Bacteriophage.**—It can be isolated from faeces pus, blood and other sources. Ordinarily faeces are made use of. The faeces are emulsified and incubated for 24 hours, then strained to remove

the coarser particles, finally being passed through a fine laboratory filter. The resulting filtrate contains the Bacteriophage.

**Demonstration and testing.**—The filtrate containing bacteriophage is added in varying dilutions to broth tubes sown with particular micro-organisms, control tubes being kept as a check. If the organism employed is one for which bacteriophage has an affinity, growth will be arrested in the tubes concerned. It can also be shown that the original bacteriophage added to such tubes has undergone an increase in quantity and in virulence during this time. Repeated passage in this manner results in a strain which may prove to be active in dilutions as high as 1 in 100,000,000,000,000.

**Bacteriophage as an immunising and therapeutic agent.**—It has been satisfactorily demonstrated that it can be used as an immunising agent in bacillary dysentery of lambs (Daubney and Gollaway), in Hæmorrhagic Septicæmia of Buffaloes (Barbone), and Fowl Typhoid (D'Herelle).

D'Herelle in Indo-China successfully immunised 12,000 buffaloes with bacteriophage during an epizootic attaining a mortality of 100 per cent. Vaccinations were continued after this epizootic and the mortality was reduced to nil, and it appears as if the focus of the disease in Indo-China has died out.

As a therapeutic agent it has been used extensively in human diseases with **considerable** success.

# NOTES.

## NOTES.

## PART II.

## THE MICROSCOPE.

**Purposes.**—The use of the microscope, so far as we are here concerned, consists of the examination of blood, pus, or other material for the presence of micro-organisms or other minute bodies.

**Description.**—For purposes of description the microscope may be divided into three parts:—

(*a*) The base on which the instrument rests.

(*b*) The tube containing the lenses, with its attachments.

(*c*) The stage for the reception of the objects to be examined and its attachments for the control of light.

(*a*) **The base,** of metal is made sufficiently heavy to maintain the microscope in a stable condition of equilibrium during use; it may be either of a tripod or horse-shoe shape.

(*b*) Working on the base by means of a pivot is an upright which maintains **the tube,** the connection between the two being a rack and pinion moved by means of screws so that the tube is moveable on the upright. This is called the **coarse adjustment.** There is another means of moving the tube known as the **Fine Adjustment.** This is worked by means of a screw in a horizontal position and moves the pillar or upright up and down.

The tube is a hollow cylinder into the top end of which fits the **eye-piece** and into the lower end the **objective.** The lower end is usually provided with an apparatus known as a **triple nose-piece,** by means of which three objectives can be screwed on, and whichever is wanted is brought into position by turning round the nose-piece, thus saving the bother of continually screwing and unscrewing the objectives. The tube itself is **double,** one tube being contained within the other and working on a telescope action. The inner one is adjusted for partial withdrawal, on which the length of the tube itself is increased and magnification of the microscope becomes larger.

(i) **Eye-piece.**—There are usually two of these, numbered respectively 1 and 2, the latter having the higher magnification power. Their purpose is to refract the diverging rays coming from the objective, so that they will reach the pupil of the eye, and at the same time magnify the image formed by the objective. In Leitz microscopes the eye-pieces range from I to V, II and IV will be used, the latter having the higher magnification.

(ii) **The objectives.**—For ordinary use three of these are sufficient, *viz.*, 2/3″, 1/6″, 1/12″, the low, medium and high powers respectively, the numbers representing the focal lengths in parts of an inch of single lenses having the same magnification as the combination of lenses in the objectives.

(*c*) **The stage.**—This is a platform situated beneath and at right angles to the tube. It is attached to the pillar or upright, and is for the reception of the specimen to be examined. Its centre is perforated

by a hole to allow of the transmission of light, this light being necessary for the illumination of the object, and carrying its image to the eye through the lenses and tube.

Beneath this hole is the apparatus for the control of the light, this consisting of a **mirror**, an **iris diaphragm** and a **condenser.**

The **mirror** is attached to the base and is revolvable in all directions. It has two surfaces, one being flat and the other concave, the latter being used with low and medium powers, and the former, with the high-power. The concave mirror, having collected the rays of light, reflects them to a point, the focal point, which is usually at the opening in the stage and causes consequently a considerable increase in intensity of light, whereas the plane mirror reflects the rays in parallel direction as it receives them.

The **iris diaphragm** is an arrangement which enlarges or decreases the size of the aperture in the stage in like manner as the pupil of the eye is enlarged or made smaller by the iris. It controls the amount of light admitted.

The **condenser,** of the Abbé pattern.—This is a system of lenses situated beneath the stage, the purpose of which is to condense light, and thus give an amply illuminated field when the illumination is otherwise deficient, but is more especially to illuminate the object with a cone of light having an angular aperture equal to that of the objective, which cannot be attained with a mirror only. Only the flat mirror should be used with the condenser, as the lenses are so placed that they collect parallel rays into an apex of a cone on the surface of the upper lens. If converging rays are used as would be reflected from a concave mirror the apex of the cone would be inside the condenser.

**Manipulation of microscope.**—Having attached the lenses and inserted the eye-piece, and assuming the specimens for examination to be ready, the first thing to be done is to place the microscope in such a position as to be favourable for light.

The sources of light are either day-light or artificial light. In the former a northern light is preferable, and in the latter a flat-wick oil lamp or an electric light bulb. When using day-light, place the microscope as nearly as possible directly before a window, and when a lamp is employed, have it on a table either in front or at the right-side of the microscope and within easy reach.

Before lighting make certain that the mirror bar is in exactly central position, and set the mirror at such an angle to the light that it will be directed upon the object (this having been put in position on the stage over the hole in the same), which can be done most quickly by observing the object directly, keeping the head at one side of the tube.

The condenser need only be used with the high power lens and can be turned out of the way when the medium and lower power lenses are being used.

**Focussing.**—The light being satisfactorily directed, the next thing to be done is to focus the objective.

With the triple nose-piece, the adjustment is such that when one objective has been focussed, the others are brought very nearly into focus by revolving them into position on the nose-piece, as required. All that is then necesary is carefully to focus with the fine adjustment.

The easiest objective to focus is the low power ($\frac{2}{3}''$). This should be first focussed, then the others can be brought into position as desired. The procedure should be as follows :—Lower the head to the level of the stage and watching the front of the objective, lower the tube, by means of the coarse adjustments, to about $\frac{1}{4}''$ from the object. Look through the eye-piece and slowly elevate by the coarse adjustment until the image is distinct. Then use the fine adjustment.

Focussing should involve no danger to the front lens of the objective or to the cover glass by their coming into contact.

When using the high power, see that the condenser is in position and its upper lens on level with the upper surface of the stage.

The diaphragm as a rule should be of the same aperture as the size of the front lens in the objective.

Light when passing through substances of different density is bent, consequently light passing through a glass slide and cover glass into the layer of air between these and the objective is bent, and there is a loss of definition. This is unavoidable with medium and low powers, but the amount of loss is hardly appreciable, and it is impossible, on account of the distance between the objective and the cover glass, to interpose anything which would reduce this loss. On the other hand, when the high power objective is being used, this loss of definition would be quite marked, but by reason of the proximity of the objective to the cover glass, it is possible to introduce something to prevent this, and the substance used is clarified specially prepared cedar wood oil.

The reason for its employment is that its refractive or bending properties are almost identical with those of crown glass, and cover glass has very nearly the same properties as crown glass. Thus, as the medium which the light passes through, between the object and the objective is practically homogenous, there is very little bending, and the loss or definition extremely small.

**To apply the cedar wood oil.**—After the stopper has been withdrawn from the bottle, allow the oil to run down the rod (which is attached to the stopper) until the last natural drop has separated from it, and apply the remainder or less than a drop to the objective. It is important that the oil should be free from dust or bubbles.

Do not swing the high power with the oil on it into position or it will smear off along the cover glass, but having applied the oil, raise the objective by the coarse adjustment, then swing into position, and lower until the oil comes in contact with the cover glass, then focus.

After use, the slide and the objective should invariably be cleaned. The fluid may be removed by a moist piece of soft linen, then cleaned with a dry piece. Chamois skin is not suitable, as it does not absorb the oil.

If oil has been allowed to dry on the objective it can be removed by applying more oil, and leaving for a short time, when it will become dissolved and the whole can be removed as above; or a little xylol will speedily remove it, but requires care, as xylol dissolves the cement keeping the lenses in position.

## THE PREPARATION OF MATERIAL FOR LABORATORY EXAMINATION.

In almost every case where a laboratory examination is to be carried out, it should be made as soon after the material is removed from the body as possible. There should therefore always be the least possible delay in forwarding such specimens to the laboratory.

In preparing specimens for laboratory examination it is very important that all reasonable precautions should be taken to prevent outside air, or other contamination.

The material to be supplied and the method of taking depend on the following:—

(1) The disease in question, *e.g.*, blood smears are indicated in Surra or Anthrax, pus swabs in Epizootic lymphangitis, etc., etc.

(2) The apparatus at hand—Laboratories can always supply cleaned microscopic slides and sterile swabs, and Veterinary Hospitals should always hold a supply of these.

(3) The laboratory procedure indiciated, *e.g.*, if agglutination tests are to be carried out, blood or serum must be supplied.

(4) The time elapsing before laboratory examination can be made, *e.g.*, it is useless sending samples of faeces long railway journeys in hot countries.

**Particulars required.**—After taking a specimen the following notes should be made on the spot, for completion of the *pro forma* which will be submitted, in duplicate, with each specimen sent to the Laboratory. A specimen copy of the *pro forma* is given in appendix 'B'.

1. Hour and date.
2. Class of animal.
3. Age and description.
4. Unit number, if any.
5. Unit.
6. Place.
7. Condition of animal.
8. If dead, date and hour of death.
9. Specimen supplied.
10. Preservatives used.
11. Diagnosis and short history of the case.
12. Name, rank and address of sender.

Similarly each slide, tube, jar, etc., should be labelled on the spot with the animal's number and the date the specimen was taken.

Where a number of specimens are being taken at the same time it is particularly important that the above procedure shall be followed, in order that mixing and confusion shall not result.

**Despatching.**—Post Office regulations regarding despatching of morbid material must be complied with.

All specimens should be checked, and where necessary re-labelled before being packed up. Packing must be secure to prevent damage or breakage, and this is particularly important when dangerous diseases (such as Anthrax and Glanders) are being dealt with. A packing note should be included to show the nature of the specimen, and name and address of sender. In addition, where dangerous conditions are involved, a warning note should be included in the outer wrapper of the package.

The actual particulars of the specimen should as a rule be posted separately and at the same time. This obviates the risk of soiling such particulars through leakage or breakage.

**Glass Slides.**—Special slide boxes should be made use of when available. In their absence the following method of dealing with slides is recommended :—

Place the slides back to back (*i.e.*, clean surfaces together) and wrap in paper.

Place the slides in a suitable box or tin and pack loosely with cotton wool or paper rolled into small balls.

Enclose the packing note, wrap up, tie and seal.

**Test tubes containing swabs.**—These should be securely stoppered and packed up in the special containers supplied for them. In the absence of these wood containers, tubes should be wrapped separately in corrugated paper. On account of their fragile nature it is generally better to make use of bottles if proper holders are not available to take tubes.

**Liquids.**—May be sent in test tubes or bottles. The former are preferable if proper containers are available. If bottles are used the neck and stopper should be wrapped round with a bandage soaked in plaster of Paris or securely sealed with sealing wax.

**Tissues.**—First cut open the tissue and soak in 5 per cent. formalin in normal saline solution. Then wrap up the tissue in cotton wool soaked in the same preservative. Again wrap the whole with jaconet and tie up securely, pack round with more cotton wool or tow, and place in a suitable tin, taking care that jostling will not take place during transit. If tissues are sent in containers of fluid formal saline, it is important that sufficient preservative be added. The volume of preservative should be at least 12 times that of the tissue sent. Composition of formal saline : 40% Formaldehyde—10 parts and Normal Saline 90 parts.

Any tissues intended for animal test inoculation should be preserved in 50 per cent. glycerine with water.

Materials from suspected cases of poisoning and Equine Encephalomyelitis will be collected and despatched as laid down in appendix 'C'.

## Blood.

**Blood films.**—The glass slides must be kept absolutely clean and free from grease. Rubbing with a soft cloth is not sufficient. First wash by rinsing in water, drain and then dry. Place the slides in 5 per cent. hydrochloric acid in alcohol for a few hours, then rinse in running water. Afterwards they are best preserved in absolute alcohol in a clean stoppered bottle. When required they should be taken up with forceps and the alcohol burnt off by passing through a flame.

The object to be arrived at in the preparation of blood films is to obtain films in which the red corpuscles will be distributed in a thin even film, one layer thick.

The best place from which to obtain blood for making films is the tip of the ear, and the method of procedure recommended is as follows—

1. Remove the hair from the ear at the tip.
2. Wipe away all loose hair and dirt from the clipped area with a piece of dry cotton wool. Swab with ether or methylated spirit and allow to dry.
3. Make a small incision in the edge of the ear with a clean scalpel or scissors.

If blood does not appear, " milk " the ear from the base to the apex.

4. When blood appears bring the flat surface of a slide, about three quarters of an inch from one end, in contact with the exuding blood allowing blood to adhere to it, or transfer a small quantity of blood from the ear to the same place on the flat surface of the slide with the corner of a second slide. The important point to remember is that the quantity of blood must be small. If it is too large the excess can be got rid of by shaking the slide and throwing off some of the blood.
5. Take another clean slide with straight smooth edges at its ends and place one end on the slide on which the drop of blood rests, just in front of the drop. Hold the upper slide firmly at an angle of 45° and place the lower slide on a flat surface such as a table.

Now bring the lower end of the upper slide into contact with the drop of blood, which spreads by capillary attraction gradually along the edge of the slide. When the drop has spread nearly to the outsides of the edge the upper slide should be propelled forwards at a uniform pace bringing along with it by capillary attraction the drop of blood and leaving behind a thin uniform film of blood on the surface of the lower slide.

When making films in a tropical country it is advisable to protect the slides from the direct rays of the sun which cause too rapid drying of the blood and the consequent formation of artefacts.

6. Now dry the film by waving it about in the air and do not fix in any way.
7. A second slide should be prepared in a similar manner.
8. To avoid the possible confusion of slides when several are prepared at the same time, they should be marked with a serial number, or the animal's number, with a grease pencil; or the number written

on the thick portion of the film with an ordinary lead pencil, or the blood scratched away with a pin or the point of a scalpel.

9. Place the films back to back (*i.e.*, clean surface together) and wrap in paper.

10. Write the particulars of the case on the paper wrapper.

In the case of Anthrax, Piroplasmosis. Trypanosomiasis, Hæmorrhagic Septicæmia, etc., blood itself is not a good thing to send. In most cases either a sterile swab soaked in blood, a slide, or serum can be substituted according to requirements. If blood is to be supplied it must be collected under sterile precautions, defibrinated, and then passed into either a Wright's blood capsule or a sterile bottle, or sterile pipette.

**Blood from carcases suspected to have died of Anthrax.**—Frequently Anthrax bacilli are too scarce to be detected by microscopic examination of blood films and swabs should be inoculated with the blood of all animals suspected to have died of Anthrax, so that diagnosis by cultural methods may be carried out if necessary. The material should be obtained as soon as possible, and the method of procedure recommended is as follows:—

1. Pour a small quantity of methylated spirit, absolute alcohol, petrol or paraffin, on the base of the uppermost ear.

2. Set a light to the liquid, and allow it to burn out.

3. If a sterile scalpel is not available flood one with one of the above-mentioned liquids and set it alight.

4. When the flame has died out, make a small incision through the skin and vessels at the base of the ear.

5. Grasp the tip of the ear in the left hand and exert sufficient traction to open the wound.

6. When blood appears insert the swab, allow it to soak up some of the blood, withdraw and return it to the test tube.

7 Insert a sufficient quantity of cotton wool into the wound to form a tight plug.

8. Wrap a moderate quantity of cotton wool or tow lightly round the base of the ear and tie it securely with string or wire.

In cases of Anthrax, especially in the horse, when there is an oedematous swelling at the throat or other part of the body, a swab should be prepared from this fluid in the same manner as described above.

### Serum.

Blood is obtained from a vein. The area of operation is shaved, cleaned up and the vein raised in the usual manner.

A Wright's Blood Capsule is made use of.

The ends are broken off and the whole heated in the flame of a spirit lamp. When cool one end of the pipette is immersed in a droplet of the blood, or if necessary even into the vein itself. Blood will flow up into the pipette by capillary attraction. If the flow is not good cotton wool soaked in ether and pressed on the capsule will cause the blood to flow up.

When sufficient blood has been collected the capsule is withdrawn ınd the far end warmed in flame. When sealed up the air inside at his end cools and by contracting draws up the blood from the other nd into the middle of the capsule. The other end is now sealed.

In sealing these capsules or pipettes care should be taken to form he end into a bead and not into a point. The former will enable the apsule to travel without breaking.

Clotting of the blood takes place within and the serum is expressed nd used later as required.

Serum is required chiefly for the Agglutination, Precipitin, and Complement Fixation Tests.

Blood is collected from the jugular vein under sterile precautions, ısing a fairly large bore hypodermic needle such as an Intravenous njection needle, direct into sterile bottles. Preferably a separate needle hould be employed for each animal, or the needle thoroughly washed ut with antiseptic solution between each bleeding. It is advisable ot to fill the bottle more than half full. Carefully avoiding shaking n transportation bottles are placed on their sides at an angle of 30°, nd blood allowed to clot at room temperature for a few hours. The xpressed serum will be found easily accessible in this position, and s sucked up into a sterile pipette, the ends of the latter being sealed or despatch. When pipette samples from several animals are sent they hould be labelled. As pipettes often arrive broken, it is advisable hat several from each animal should be sent containing as much serum s possible. Serum must be clear, as serum which is haemolysed is of o value. A few drops of toluol may be added as a preservative.

## Pus.

**Pus smears.**—The object to be arrived at in the preparation of pus mears is to obtain the material in a thin even smear without lumps r blank areas. Provided that care is exercised this object can be ttained by spreading the pus on a slide with a scalpel. An alternative nd better method is as follows:—

1. Transfer a small quantity of discharge from the lesion to the entre of the flat surface of one slide with a scalpel or other suitable nstrument.

2. Lay a second slide on top of the first one so that the pus is andwiched between the two.

3. Spread the pus by gently pressing the two slides together.

4. Take the opposite ends of the two slides in each hand and lide them apart, being careful not to lift the one from the other, s this will cause the formation of lumps and bubbles. If a thin even mear is not obtained the first time the slides are shown apart, place hem together again, gently press them together and again draw hem apart.

5. Dry the films in the air.

6. Label with a grease pencil, or by writing on the smear with scalpel.

7. Place the slides back to back and wrap in paper.
8. Write the particulars of the case on the paper wrapper.

**Swabs of discharges from open wounds:—**

1. The material should be obtained in the morning before the wound has been dressed.
2. With a clean piece of cotton wool wipe away all discharge adhering to the eternal orifice of the wound and squeeze the wound to expel as much pus as possible.
3. If the wound is large, have the orifice held open and then insert a sterile swab into the base of the wound, and in so doing avoid touching the outside edges of the wound.
4. Give the swab handle a half turn, then withdraw it and return it to the test tube.
5. Label the tube and replace it in the test tube carrier.

**Swabs of the contents of closed abscesses or pustules:—**

1. Remove, with scissors, all hair covering the lesion.
2. Clean the skin over the lesion with dry cotton wool.
3. Wipe it with cotton wool soaked in tincture of iodine.
4. Open the cavity with a sterile scalpel.
5. Swabs should be taken from (*a*) the wall of the abscess, and (*b*) the contents of the abscess.

The succeeding steps are the same as in the preparation of swabs of discharges from open wounds.

When taking swabs from pustules, etc., of dead animals the overlying skin should be more effectively sterilised by cauterising it with a red hot spatula or other suitable instrument.

## Peritoneal Fluid.

When the abdominal cavity is opened, separate the bowels and insert the swab between the folds into the fluid, taking care to avoid touching any part of the viscera which have been handled or are likely to have been contaminated. The succeeding steps in the process are the same as 4 and 5 above (Wound discharges).

## Tissue.

**Smears.**—Cut out a small piece of the organ, or bisect the nodule or lesion, and holding it in a pair of forceps, rub the cut surface of the tissue on the middle third of the slide, leaving the ends blank. If only a few cells adhere to the slide, scrape the cut surface with a scalpel, and repeat the spreading process. Caseating and all similar material from lesions can be spread in the manner described for pus.

Do not stain or fix.

Dry, label and wrap the slides in paper.

**Rabies.**—See chapter on Rabies.

### Internal Parasites.

Collect, wash in water and immerse in warm 70 per cent. alcohol (Absolute alcohol or Methylated spirit 7 parts, water 3 parts). This kills the parasites, straightens them, and preserves them.

### External Parasites.

Macroscopic parasites may be collected direct, by means of the fingers or forceps. They should be placed in a small bottle and stoppered. Preservatives are not required.

**The taking of Mange scrapings for the microscopical diagnosis of Mange:—**

1. In very slightly affected cases, the material should be taken from the area where the animal shows most itchiness on being scratched.

2. In cases showing definite skin lesions, select an area where these are most marked.

3. If there is much hair present, remove as much of it as possible with scissors and discard it.

4. Moisten the selected area with a 10 per cent. solution of caustic potash. If this is not available ordinary plain water may be used.

5. Scrape the area with a blunt scalpel—a sharp knife has a tendency to cut the skin rather than to scrape it.

6. When the lesions are small and scattered, scrape several of them until sufficient material has been obtained.

7. If the material is being collected in an envelope fold back the flap and place the inner surface of it against the animal just below the area being scraped, holding the envelope open by inserting two or more fingers into it. All loose scales, scurf, hair, etc., will then fall directly into the envelope. A large test tube may be used to collect the material instead of an envelope.

8. In all cases scraping should be carried out until blood exudes from the scraped area, as it is only by reaching the blood that the inclusion of all varieties of parasites can be ensured.

9. Each time a small quantity of blood has collected on the knife, lightly scrape the hair on some other part of the animal with the knife to cover the blood with dirt and loose hair. Wipe the knife on the inner and upper portion of the envelope and then touch it with the scalpel so that it falls among the scrapings previously collected. By doing this one prevents the blood adhering to the paper from which position it is difficult and often impossible to separate it completely for examination.

10. As much material as will cover a Rupee piece should be taken from all suspected cases of mange. The difficulty experienced in establishing the presence of a mange acarus is frequently due to the fact that insufficient material is examined and also to the fact that only superficial material is obtained when the scraping is collected.

## Milk.

Draw from the udder direct by means of a milking tube (syphon). This must be done aseptically.

The milk is to be received into a sterile tube.

Preservation is important. Ice is the best and a thermos flask packed with ice may be used to take the tube of milk, ·5 per cent. to 1 per cent. boric acid can also be used or a small quantity of 4 per cent. formalin. The latter, however, renders the sample unfit for cultivation experiments later on.

Milk is required for bacterial examination, estimation of bacterial content, dirt, detecting the presence of the tubercle bacillus, etc.

## Faeces.

Receive into a sterile wide mouth container with stopper. No preservatives are required. The specimen should be forwarded without delay. Incubation should be avoided as it will lead to the development of embryos in eggs. Parasitic ova may be differentiated by species, but embryonic forms only with difficulty.

Another method is to pack the sample in lint and greased paper inside a large box.

Fæces are required for detecting the presence of parasitic ova and parasites, certain bacteria, etc., etc. In dealing with Johne's disease rectal scrapings are preferable.

**Staining.**—Staining methods may be divided into three classes:—

(1) Simple staining.
(2) Differential staining.
(3) Special staining.

1. **Simple staining.**—In simple staining, a single stain only is used. All that is necessary is to fix the preparation, flood the slide with the strain by means of a pipette or glass rod, allow it to act for 3—10 minutes, wash with water, and dry by waving about in the air, or passing the slide gently over the flame of a spirit lamp.

In most cases there are many elements present in the film, or smear, in addition to bacteria, *e.g.*, leucocytes, tissues, etc., and these elements with a simple stain, take up the stain also, giving a more or less uniform colour to the whole preparation so that simple staining is generally reserved for examining films containing bacteria only, *e.g.*, a preparation made direct from a culture.

2. **Differential staining.**—In this method two or three stains are employed either in combination, or one after the other. The employment of differential staining methods resutls in a preparation in which the different bacteria or tissues stain different colours, *e.g.*, Leishman's stain imparts a faint blue colour to protoplasm and a red colour to the cromatin in leucocytes or protozoa.

3. **Special staining.**—Certain stains have a special action with certain tissues, bacteria, etc., and are made use of for detection or

diagnosis, *e.g.*, the McFadyean method of staining for Anthrax bacilli, gives a pinkish colouration to films where these bacilli are present.

The procedure required for differential and special, staining is given where applicable, under the description of the various diseases in this handbook.

Stains are supplied to veterinary hospitals in tabloid form in a small phial surrounded by a booklet containing full instructions as to the preparation and use of the stain.

**Mounting.**—A small drop of thick solution of canada balsam in xylol is placed in the centre of a slide and the cover glass, surface downwards, deposited on the balsam, which then spreads out, and finally extends over the whole under surface of the cover glass.

For the examination of moist films, *e.g.*, in searching for trypanosomes, the drop of blood is placed on a slide, and a cover glass placed on top at once, and the specimen examined immediately; or if the drop is taken on a cover glass, the cover glass is at once dropped on to a slide, the blood spreading out between the two.

**Table of Magnifications**

of objectives in combination with eye-pieces. Tube length 170 mm.

**Lietz's Microscopes.**

| Objectives. | Eye-pieces. | | | | Use. |
|---|---|---|---|---|---|
| | I | II | III | IV | |
| Low power objective 3 (Leitz) or $\frac{2}{3}''$ . . | 60 | 70 | 80 | 105 | For acari. |
| Medium power objective 6 (Leitz) or $\frac{1}{6}''$ . . | 255 | 300 | 350 | 460 | For detection of Trypanosomes in fresh blood. |
| Homogenous oil immersion 1/12″ objective . . | 555 | 650 | 800 | 1,000 | All bacteriological fine work. |

# NOTES.

# NOTES.

## PART III.

## GENERAL MEASURES FOR DEALING WITH OUTBREAKS OF CONTAGIOUS AND INFECTIOUS DISEASES.

On contagious disease being diagnosed, the following broad principles of action should be observed:—

1. Removal of animals from lines or standings.
2. Isolation, or segregation, adopting group system of (*a*) affected (*b*) suspected (*c*) apparently healthy.
3. Destruction of affected, if necessary.
4. Disposal of carcases.
5. Disinfection.
6. Reports.
7. Periodical inspection.
8. Working isolation.
9. Preventive inoculation.

### 1. Removal from lines or standings.

Whether all animals should vacate their lines, or only the affected and those in contact (suspected), depends on the infectivity of the particular disease, the extent to which contagion has spread, or the manner in which the disease may be contracted. With a highly infective disease like Rinderpest, where the ground is readily infected by alvine discharges, standings should be changed, infected ground being avoided; in Glanders if the outbreak is not severe, only affected and incontacts need be removed. **In every case it is essential to remove affected and incontacts.**

### 2. Isolation—Segregation.

(*Vide* paragraph 847, Regulations for the Army in India. Instructions.)

The animals should be divided into three groups: the affected, suspected and healthy. The suspected should include all those which have been in contact with the diseased, although they might not be showing any suspicious symptoms at the time. Attendants, watering and feeding arrangements and all gear should be included in this separation. The distance between the different groups should be sufficiently far to prevent any chance of communication between them. Contagion can be carried by attendants, flies, vermin, poultry, dogs, etc. When military considerations restrict the space at disposal fencing of some sort should enclose each group, and if there is a prevailing wind the healthy should be placed to windward and the suspected and affected to leeward in the order named.

Once an animal is placed in the affected group it should remain there till the outbreak is over and on no account be replaced among the healthy, although it may have been moved in error in the first instance.

### 3. Destruction of affected.

Regulations enjoin that when any case of contagious or infectious disease constitutes a public danger, the animal, whether public property or the property of any person in military service, may be destroyed on the written opinion of a Veterinary Officer under the order of the O. C. the station. (Paragraph 845, Regulations for the Army in India. Instructions.)

Cases of a virulent rapidly infective disease attended with great mortality, such as Rinderpest, may be destroyed to prevent spread of infection: cases of incurable disease such as Glanders-Farcy, or severe Epizootic lymphangitis should invariably be destroyed. This will be more fully dealt with under the description of individual contagious diseases.

Great care should be exercised in the shedding of blood in cases of contagious disease where the blood is highly infective, or where on its exposure to air spores are formed as in anthrax.

### 4. Disposal of carcases.

In all cases where discharges are infective, the natural orifices should invariably be plugged with tow or cotton wool soaked in some strong disinfectant when carcases are removed from the place of death or destruction to the place of burial or incineration. This should particularly be done if public roads have to be traversed. Carcases should be conveyed in carts, and should not be drawn along the ground.

In disposing of carcases, the life history of the causal agent should be considered. In all diseases in which there is a probability of spore formation as in anthrax, or where the causal agent may lead a saprophytic existence, or is especially resistant as in Epizootic lymphangitis, the carcases should be destroyed by fire. For this purpose one or more incinerators of a pattern not wasteful of fuel should be built in every military station, or failing this as on field service, a trench dug in the ground in the shape of a + should be used. The trench should be 7 feet long, about 15 inches wide, and 18 inches deep in the centre where the two cross arms meet, and becoming shallower, as they rise to the surface of the ground. The earth should be thrown into the angles formed by the trench, and on this should be placed two stout pieces of iron, *e.g.*, ordinary railroad rail 3 feet long for the carcase to rest on. If the carcase is eviscerated and the limbs are removed, it burns more readily. In India 13 maunds of wood and one-third of a gallon of kerosene oil are allowed for burning of each carcase of horses, camels and bullocks, and 8 maunds of wood and the same amount of kerosene oil for mules and ponies.

If anthrax carcases cannot be burned they should be buried **whole** deeply. Carcases of animals which have died from or been destroyed for Glanders or Rinderpest may either be burned or buried. Surra carcases may be burned or buried during the surra season, but need

only be buried at other times of year. In all cases excepting anthrax the skin should be slashed and the carcase covered with some disinfectant to prevent it being dug up and used. Carcases should be covered with 5 feet of earth in burial, particularly in anthrax. Earth soiled with blood or discharge at the place of incineration or burial, say at *post mortem* examination or evisceration, should also be burned or buried. Burial should be effected, in unfrequented ground, away from water-supply; and in the case of anthrax the ground should be fenced in and marked with a printed notice "anthrax infected ground".

On field service the practice of leaving carcases on the road side along a line of march should be prohibited. Arrangements should be made for burial, or if this is not feasible, they should be taken **at least** 1,000 yards away from the road.

## 5. Disinfection.

To apply intelligently any system of disinfection two main points should be borne in mind, *viz.*:—

(1) The life history of the microbe causing each disease, and its resistance, to external influences such as light, heat, desiccation and chemical agents.

(2) To direct measures particularly against the dangerous elements of the disease, *e.g.*, the discharge from the nose in Glanders, the virulent blood and alvine discharge in Rinderpest, the blood or sero-sanguineous discharges of Anthrax, the pus of Epizootic lymphangitis, the saliva and serous discharge at the coronet in foot and mouth disease, and so on.

With regard to (1) it is known that the virus of Rinderpest will live barely three days outside the animal body; that the virus of foot and mouth disease though persistent and extremely easily carried, has a vitality about eighteen days outside the body; that the virus of Rabies is inert after fifteen days drying; that the organism of Surra is dead twenty-four hours after the death of the host; that the bacillus of Glanders in discharge or putrefying material in the open air may remain active for 14 to 24 days, and in closed stables may even be virulent up to 4 months; that the bacillus of anthrax can live as a saprophyte developing spores which can retain their vitality unimpaired for years, and that the organism of Epizootic lymphangitis has an indefinite vitality outside the body and may even be a saprophyte.

In considering resistance to external influence it should be borne in mind that micro-organisms are for the most part of the vegetable kingdom. Agencies, therefore, which best destroy or hinder ordinary plant life, will prove effective against microbes.

**Light** has an injurious effect on all bacteria, exposure to air or sunlight preventing their growth, and even development of spores.

**Fire** acts as a total destructor, and for the disposal of infective carcases, manure, bedding, and the disinfection of the surface of standings, it is most convenient and should always be resorted to whenever possible.

**Boiling water,** for the same reason should be made use of. **Steam,** which should always be saturated and not superheated, is most destructive, and is of great value in the disinfection of railway trucks, etc. To ensure proper disinfection by **moist heat** saturated steam at 239° to 230° F. should be allowed to act for at least 15 minutes. All pathogenic germs are killed by **Moist heat** at 149° F., or even less, acting for ten minutes. Exposure to steam will kill the spores of Anthrax (the most resistant of all spores) in three minutes, and boiling will kill them in five minutes. **Dry heated air** is not so effective, and has little power of penetration. With dry heated air, a temperature of 248° to 262° F. acting for at least one and a half hours is required to kill most pathogenic organisms, and 284° F. for three hours to kill the spores of Anthrax. It is therefore of little value as a disinfectant.

**Cold** is tolerated by most micro-organisms.

**Desiccation** is inimical to some microbes, but under it others retain their vitality. Thorough drying and action of air and sunlight is, however, a necessary condition in the final stage of disinfection. Moisture favours the growth of micro-organisms as it does that of plants.

**Chemical agents** are very destructive by virtue of their poisonous action on protoplasm, and form the basis of disinfection as usually practised. **Acids** are very complete in their action but they are corrosive and moreover expensive. An Anthrax carcase treated with sulphuric acid or sand mixed with sulphuric acid on burial has been recommended. **Gases** are of little value in India where stables are more or less open. Fumigation is little practised now for the reason that microbes do not float in the air, as was formerly supposed unless in a dry condition, but adhere to walls fittings, floors, etc., where direct application of disinfectants more satisfactorily **and surely deals with them.**

**Disinfectants.**—For practical purposes of disinfection in veterinary service in India, the following scale of Disinfectants is approved as sufficient, and need only be considered:—

Corrosive sublimate.

Carbolic Acid.

Chlorinated Lime.

Quicklime.

Common Salt.

**Corrosive sublimate** (perchloride of mercury) heads the list of all disinfectants for certainty and reliability. A solution of 1 in 1,000

will destroy the bacilli of Glanders and Anthrax in ten seconds, and the spores of the latter in ten minutes. It has the disadvantage of forming compounds with albuminoid substances, but this is greatly prevented by addition of common salt up to five times the quantity of perchloride. It is used in solutions from 1 in 400 to 1 in 1,000 in water.

**Carbolic acid** is most useful as a disinfectant and is stable in the presence of organic matter. It is not of much value in the destruction of spores. Its efficiency is increased greatly by addition of common salt up to saturation. A 5 per cent. solution made with boiling water should be used. The ordinary impure carbolic acid will only form a 3·5 per cent. solution; pure crystals form a 8·6 per cent. solution. Carbolic powders with a lime basis are useless.

**Chlorinated lime** (improperly styled "Chloride of lime") is cheap and of great value for the disinfection of floors, standings, drains, walls, railway trucks and for mixing with fæces and discharges. It may be used either as powder, being sprinkled evenly over standings, floors, etc., or it may be used in solution of ½ lb. or 1 lb. of Chlorinated Lime to 1 gallon of water, the latter forming a 10 per cent. solution. To be of any value, Chlorinated Lime should be fresh. Its value as a disinfectant may be judged by it being very irritating to the eyes when brought near the face. Solutions should be freshly made, and applied at least three times when disinfection of railway trucks, walls, etc., is being carried out.

**Quick lime** is only of use in a **caustic** state. It should be freshly slaked at time of use, sprinkled then as powder over standings, floors, drains, etc., and mixed with the earth of standings to be disinfected. As lime wash made by the addition of water to freshly slaked lime, it is used for limewashing walls, but it should be remembered that lime is inefficient against the more resistant micro-organisms, and that limewashing is not sufficient protection. Whitewash, which is limewash without caustic action, is useless as a disinfectant.

**Common salt** is useful for preserving and assisting the action of both Perchloride of Mercury and Carbolic Acid.

## Routine of disinfection.

Bearing in mind, therefore, the life history of the causal organism of the particular contagious disease, its vitality and resistance to outside influences both physical and chemical, and the means and materials for disinfection at our disposal, it is necessary to adopt a definite line of action in disinfection, particularly directing operations against what is the most dangerous article in contagion, or what from the nature of the disease is most likely to be contaminated.

The following Routine is given for general guidance:—

| To be disinfected. | Procedure. |
| --- | --- |
| 1. Paved floors and permanent standings. | Burn litter or straw over surface. Wash with 5 per cent. solution Carbolic Acid or 10 per cent. Chlorinated lime. Repeat two or three times, the last or two last applications being 1 in 400—1,000 Solution of Corrosive Sublimate: expose to action of air and sunlight if possible. Boiling water should be used for making these solutions and common salt added to those of Carbolic Acid and Corrosive Sublimate. |
| 2. Earth standings . | Of standings which have been occupied by infected animals, and of standings on either side that are likely to have become contaminated, scrape off the surface layer of earth. Cover the standings with straw or bedding, mix the scrapings loosely with it and carefully burn the whole, seeing that scrapings and surface are well charged. Due care must be exercised in thatched stables. Then dig up standings to a depth of one foot, mix the dug up earth with a 10 per cent. solution of Chlorinated Lime or Milk of Lime (1 part slaked lime, 2 parts water), remove from lines to a convenient place, where for safety it may be spread out in a thin layer and again burned.<br>Sprinkle dug out standings with Chlorinated Lime or freshly made quicklime, and expose to action of air and sunlight for a fortnight. After that time relay the standings with fresh earth in which a little quicklime is mixed. |
| 3. Mangers . , . | If of earth, destroy, and treat exactly as described under heading "Earth Standings", care being taken to destroy any food remaining in mangers.<br>If of iron, expose to fire. This can be effected by straw, but is much more conveniently and effectively done by a petrol or kerosene (preferably petrol) braizers' lamp. If this can be obtained, it will be found of undoubted value, and a cheap method. |

| To be disinfected. | Procedure. |
|---|---|
| | Then by means of a brush, scrub with boiling water and soap, preferably soft soap, and afterwards apply a 5 per cent. solution of Carbolic Acid or 1 in 1,000 (or stronger as the case may warrant) solution of Corrosive Sublimate. Repeat the application three times, at most every third day. In all greasy conditions, Carbolic acid should have preference to Corrosive Sublimate, at least for first application of disinfectants. |
| 4. Walls . . . | Sweep first with damp brush or cloth, and afterwards scrape well. Burn sweepings and scrapings carefully. Then wash with a 10 per cent. Solution of Chlorinated Lime, a 5 per cent. Solution of Carbolic Acid, or 1 in 1,000 Corrosive Sublimate Solution. Repeat at intervals up to three times, and expose to air and direct sunlight if possible for a fortnight. Solutions are best applied with a spray. In wall disinfection take care not to overlook cracks, crannies, corners, ledges, saddle racks and other places difficult of access. Walls in front of mangers, should have particular attention. After a fortnight's exposure, walls may be re-limewashed, coloured or tarred accordingly to custom. A little sulphate of copper may be added to lime or colour wash. |
| 5. Woodwork, Doors, Windows. | Scrape well and burn scrapings. Then scrub well with boiling water and soft soap, afterwards applying disinfectant solution. As doors particularly are apt to be very greasy, use 10 per cent. solution of Chlorinated Lime or 5 per cent. Carbolic Acid first, and solution of Corrosive Sublimate afterwards. Repeat applications at intervals as previously described, thoroughly wetting all cracks in the woodwork. Repaint doors and windows after a fortnight. |
| 6. Water troughs . | To the water contained in the trough add sufficient disinfectant to bring the resulting dilution to the required strength, *i.e.*, 1,500 Corrosive sublimate or 10 per cent. |

| To be disinfected. | Procedure. |
|---|---|
| | Chlorinated lime. After 24 hours the trough should be drained, allowed to dry and then treated with a blow lamp or refuse hay or straw burnt in it. After that it should be thoroughly scrubbed with 1 : 500 Caustic potash solution applied with a stiff brush. To remove all traces of disinfectant it should again be scrubbed with water and rinsed out. Fresh water should be allowed to fill the trough, and remain in for 24 hours, and then discarded. The trough may then be brought into use. |
| 7. Bedding, Excrements. | Destroy by fire. If excrement of affected animals under treatment cannot be at once conveniently burned it should be mixed with Chlorinated Lime or quicklime, particularly if it has to be removed any distance from the immediate vicinity of affected animals for burning. Care should be exercised in its removal. Corrosive sublimate is not suitable for the disinfection of fæces. |
| 8. Drains . . . | If there has been contamination from infected standings or water troughs, treat exactly in the same manner as stated under heading "Standings," as drains are surface drains in India. |
| 9. Clothing and Line gear, including stable head collars, nose bags, grooming kit, buckets, picketing rope, etc. | Which of the articles of gear are to be destroyed will depend on the nature of the particular case, but as a general rule rubbers, brushes, numdahs, blankets and nose bags should be burned and such articles as are likely to have become dangerously contaminated. If blankets are not destroyed, steep in 10 per cent. solution of Chlorinated Lime or 5 per cent. solution of Carbolic Acid for 12 hours, boiling water being used for solution. Repeat at intervals of three days up to three times, and expose to sun for a fortnight; or, if available, pass through a "Thresh" disinfector.<br>Pass all iron articles through flame, and immerse in above disinfectants for several hours, or boil if convenient. |

| To be disinfected. | Procedure. |
|---|---|
| 10. Saddlery . . | Scrub all leather articles well with warm water and soft soap. Wash three times at intervals of three days with 10 per cent. solution of Chlorinated Lime, or a 5 per cent. solution of Carbolic Acid. Strip saddles for the process, and see that all interstitches and under-surfaces are thoroughly done. Dry slowly and carefully after each application lest saddles warp, and when thoroughly disinfected and dry, rub a little oil or saddle soap into the leather.<br>Treat ironwork of saddlery as stated under heading " Line gear ", etc. |
| 11. Attendants, their clothing, etc. | This is very important in view of the virus of such diseases as rinderpest and foot and mouth disease being so readily carried.<br>Attendants should be rigidly isolated as well as animals. They should not be allowed to leave the precincts of an affected group of animals.<br>They should be shoeless, and have the sleeves of their coats rolled up to their elbows, as sleeves are very apt to become contaminated in the handling of animals. Maintain a supply of hot water, a towel, soap and a 5 per cent. solution of Carbolic Acid, and instruct attendants to begin and end their day's work with washing of hands and arms, feet and legs with water and soap, afterwards using the disinfectant solution. This is to be repeated particularly after handling or dressing affected animals.<br>Change of wearing apparel should be arranged for, any soiled garments to be soaked in the above disinfectant solution for a few hours, and washed on the premises. On conclusion of outbreak give attendants at least three days' leave so that they can have a thorough cleansing before rejoining healthy unit. Arrange with S. M. O. for final disinfection of their clothing.<br>Visitors to lines should leave walking sticks behind, and before leaving should wash their hands as described above, if they have handled affected stock, and certainly |

| To be disinfected. | Procedure. |
|---|---|
| | should disinfect their boots in the disinfectant solution maintained. |
| 12. Horse-boxes and trucks. | Remove excrement, scrape sides, partitions, floors, and burn excrement and scrapings near by. Arrange with Railway Companies (such facilities usually existing at big centres) to disinfect by means of steam, thoroughly saturating each truck inside and out, particularly inside, for a quarter of an hour. Afterwards, spray with 10 per cent. solution of Chlorinated Lime, and expose to action of air and sunlight. Repeat application of Chlorinated Lime solution two or three times at intervals of two or three days.<br>Failing disinfection by steam, thoroughly scrub with boiling water, soft soap, and a 5 per cent. solution of Carbolic Acid, afterwards applying Chlorinated Lime solution as above mentioned. Trucks that have carried affected animals should not be used for at least a fortnight after first disinfection. |
| 13. Disinfection on ships. | Depends greatly on the nature of the disease and extent to which infection has spread.<br>Do not adopt any half measures, and exercise utmost vigilance to prevent spread.<br>Presuming ship is well out at sea, throw over board all possible fittings of standings of animals dead or destroyed as affected; deal similarly with excrement and fodder contaminated, or likely to be contaminated.<br>Make every use of steam for disinfection when possible, and stalls can be vacated. Thorough flushing with sea water is extremely valuable as a preventive of disease during voyage, or for thorough cleaning at end of voyage. Be careful not to spread infection by indiscriminate flushing during an outbreak.<br>Make free use of boiling water and soft soap, and the chemical disinfectants enumerated above, repeating their application. Care must be exercised in the use of Chlorinated Lime, on account of irritation to the eyes. |

Attention is drawn, in these notes, to the repetition of the application of disinfectants after intervals of several days. This practice should invariably be followed, and most certainly in contagious diseases attended with probable spore formation. The reason for this is that in the intervals of disinfection spores may germinate under favourable conditions and would be destroyed by the successive disinfections. The process may be termed **fractional sterilisation or disinfection.** It is impossible to tell how long it will take a spore to germinate, this varies according to circumstances, but for practical purposes we may fix repetition at intervals of three days.

### 6. Periodical Inspection.

Make inspections of infected units or groups daily, the apparently healthy first, then the incontacts and suspected, and lastly affected animals. While inspecting nostrils the inspector's hands should be frequently disinfected, and particularly in any doubtful case. Nostrils should not be cleaned before an inspection. Use a thermometer in every case where it may be a guide to detection of the disease. During the prevalence of contagious and infectious disease at a station all animals will, under the orders of the O. C. the Station, be examined fortnightly by a Veterinary Officer (paragraph 845, Regulations for the Army in India instructions.)

### 7. Working isolation.

Animals are sometimes placed in " working isolation ", generally when Lymphangitis Epizootica has made its appearance. " Working isolation " means that the animals work as usual with the following exceptions :—

(*a*) They are not to use the same watering trough as other animals.

(*b*) They are not to occupy the same standings or intermix with other animals.

(*c*) Frequent inspections to be made and all animals with wounds, however slight, to be isolated until the wounds have healed.

(*d*) No interchanges of attendants or equipment to be made.

### 8. Preventive inoculation and use of diagnostic agents.

The Mallein test should be the rule in outbreaks of Glanders.

The following serum or vaccine may be used in severe outbreaks of the relative diseases :—

1. Anti-anthrax.
2. Anti-rabic.
3. Anti-rinderpest.
4. Anti-blackquarter.
5. Anti-hæmorrhagic septicæmia.

These are fully dealt with in the description of each disease.

### 9. Early diagnosis.

The importance of early diagnosis in outbreaks of contagious disease cannot be exaggerated. If the initial case or cases are recognized the number of incontacts is limited and the measures outlined in the preceding paragraphs are easier to apply. It is for this reason that paragraph 52, Regulations for the Veterinary Services in India, prescribes blood examinations for all animals that have fever for more than 24 hours. The mercuric chloride test for the diagnosis of surra in camels forms a recent valuable addition to our powers to deal effectively with the disease. It will be referred to later in this book when surra is being discussed.

## NOTES.

## PART IV.

### CONTAGIOUS DISEASES IN DETAIL.

Outbreaks of the following diseases prevailing amongst military animals will be notified to military authorities as Infectious and Contagious Diseases:—

1. African Horse Sickness.
2. Actinomycosis.
3. Anthrax.
4. Bacillary Necrosis.
5. Blackquarter.
6. Botriomycosis.
7. Coccidiosis.
8. Contagious Abortion.
9. Contagious Bovine Pleuro-pneumonia.
10. Contagious Pneumonia.
11. Contagious Stomatitis.
12. Encephalo-myelitis.
13. Epizootic Lymphangitis.
14. Foot and Mouth Disease.
15. Glanders.
16. Haemorrhagic septicaemia.
17. Influenza.
18. Jhooling.
19. Johne's Disease.
20. Mange.
21. Piroplasmosis.
22. Rabies.
23. Rinderpest.
24. Strangles.
25. Tetanus.
26. Theileriasis.
27. Trypanosomiasis.
28. Tuberculosis.
29. Ulcerative Lymphangitis.
30. Variola.

### AFRICAN HORSE SICKNESS

**Note:**—This disease is unknown in India, but a severe outbreak having occurred amongst horses and mules at Aden and in the Aden Hinterland in the autumn of 1906, a short description in this handbook is considered necessary. It may be mentioned that after that outbreak, for some time, the return of horses and mules from Aden to India was prohibited. Excepting in the instance mentioned above, the disease is unknown outside Africa. It is a veritable scourge to the equine race of that country.

**Nature.**—It is a specific, highly infective disease of horses, mules and donkeys, characterized by intense vascular congestions, and profuse

exudation of liquor sanguinis into the lungs, thoracic and pericardial cavities, or the subcutaneous tissues of the head and neck. It is attended with great mortality.

**Symptoms and diagnosis.**—There are two forms ,of the disease *viz.*, "Dun-kop" or the "Pulmonary" form, and "Dik-kop" or the "Swollenhead" form.

In the "Dun-kop" or pulmonary form, symptoms are, as a rule, comparatively absent. The animal appears in perfect health and vigour, and within an hour respiration becomes very accelerated and dyspnoeic, the animal staggers about, falls down, ejects a mass of white froth from the nostrils and mouth, and dies in a few minutes. In other cases the disease is not so fulminant, the animal lives several days, and there may be noticed high fever gradually increasing day by day until the end when it falls to subnormal, great prostration, hurried laborious respiration, dark red or cyanosed mucous membranes, a small, irregular, almost imperceptible pulse, a rattling in the bronchial tubes and at the lower end of the trachea attended with soft mushy cough, a clear amber-coloured discharge from the nostrils or an accumulation of white froth around the nostrils and mouth. The abundance of froth blocking the air passages causes death by suffocation. This form is very fatal.

The "Dik-kop" form is not so fatal, and the effusion of serum is located in the head and neck, which sometimes become enormously swollen. There is also present the small irregular pulse, prostration, a pinkish yellow colour of the mucous membrane, and amber trickle from the nostril and other symptoms of the severe pulmonary form in a modified degree. Sometimes the swelling is mostly confined to the tongue which presents a livid colour. To this subvariety the term "blaauw-tong" or "blue tongue" is given.

**Post-mortem appearances** are very characteristic. White froth covering the nostrils, when the carcase is lying on the ground on which it died, is almost invariably present, and it is quite diagnostic of "Horse sickness" in whatever country it is seen. Indeed no other indication is necessary for confirmation of diagnosis. The froth persists for a considerable time after death. The trachea and bronchial tubes are also filled with it. The lungs are voluminous, and the interlobular spaces are well marked with a characteristic yellow gelatinous exudate, which towards the free edges of the lungs may be half an inch thick. There is no true inflammation of the lung tissue. The pericardial sac usually contains a straw coloured and slightly blood-strained exudate. In "Dik-kop" the same bright yellow gelatinous exudate is found in the subcutaneous connective tissues of the head and neck.

The mucous membrane of the pyloric portion of the stomach is congested and port wine coloured.

**Bacteriology and infection.**—The micro-organism which produces the disease has not yet been discovered. It is one of the filterable viruses. It is so small that it passes freely through the pores of a Berkefeld filter and is not even arrested by the closest grained porcelain filters. It cannot be cultivated outside the body.

The virus has wonderful powers of resistance: horse sickness blood, dried and pounded into powder, is inert (though McFadyean found it active after six days' simple drying at 98·6°F.): there is no loss of activity on heating blood to 131°F. for ten minutes, and it is virulent after years if kept in a moist condition, as in blood, even though the material is putrefactive. Blood preserved in an ice chest in half its volume of glycerine is infective after ten years. (McFadyean).

The disease is not contagious in the ordinary sense of the word. How infection is produced is still a debatable point. A horse can be stabled alongside a case of horse sickness without contracting the disease, or a horse may occupy, without danger, a standing in which an animal has recently died from it. Considerable evidence has been accumulated to show that some biting insect is concerned in its communication, and the history of cases, combined with the habits of mosquitoes, rather point to a variety of that insect being the communicating agent. The "reservoir" of the virus is not known: an intermediary host is suspected.

Formerly (and still now by some inhabitants of South Africa) it was attributed to eating dew-laden grass, but inoculation with dew produces no ill results. The disease is not usual in stabled animals, is more frequent in animals living a life in the open, and has, moreover, a seasonal prevalence, namely, during times of rain when biting insects abound.

It occurs in zones; altitude has a protective influence; and it dies down with cold weather or frost; in fact all knowledge of the disease points to its causation through biting insect agency.

The blood is very infective, the smallest amount producing the disease on inoculation. Incubation is from 6 to 15 days.

Animals recovered from the disease are said to be "salted". They are practically immune. The term is also applied to animals that are resistant from extended residence in a bad horse sickness district.

**Immunisation.**—The serum of a horse that has recovered from horse-sickness is not a potent anti-serum, but a potent anti-serum can be produced by hyperimmunisation. The method adopted to hyperimmunise a recovered horse is by transfusion of blood from a virus producing horse at definite intervals, up to 10 litres of blood at each transfusion.

Horses are with difficulty immunised against horse-sickness, the only known safe method being a rather cumbersome one, and therefore not of much practical value. This vaccination entails the use of 2 viruses, *viz.*, an initial attenuated virus and a second highly virulent virus. The first virus is injected alone intrajugularly, and the second is injected on the 6th day together with serum at so much per Kilo body weight, the injection of serum being repeated on the 8th day. Rather large quantities of serum are required.

Horses cannot be safely immunised by the ordinary serum-simultaneous method, as too many fatalities result from the use of a strong virus, an insufficient immunity is obtained with a weak virus.

Mules can be immunised by the serum-simultaneous method, serum and virus being injected at the same time, the virus intrajugularly and the serum either intrajugularly or subcutaneously.

**How to deal with an outbreak so far as India and her Dependencies are concerned.**

1. Stamping out must be the order of the day.

2. As the disease would in all probability be first recognised by finding an animal or animals dead, with the characteristic froth at the nostrils, carefully remove carcases, after first covering them with a strong smelling disinfectant to keep off biting insects, and completely incinerate them, taking care to avoid soiling the ground.

3. Destroy any other diagnosed cases and incinerate. Treatment on no account should be adopted.

4. Stable all unaffected animals of the unit, particularly after sundown. " Chicks " will be necessary in open stables. If in camp, vacate the locality, if possible removing to high ground.

5. Keep down mosquitoes and biting insects in every conceivable way, and protect animals from their attack as circumstances dictate. " Smoke " fires made by burning damp litter are probably the most effectual single measure which can be taken to achieve this objective.

6. Units that are free must be kept well away from an affected unit.

7. Thoroughly disinfect all places occupied by diseased animals.

# NOTES.

# NOTES.

## ACTINOMYCOSIS.

*(Wooden tongue, Lumpy jaw.)*

**Nature.**—This is a specific disease affecting several species of animals, particularly cattle and man, due to a vegetable parasite commonly known as the "ray fungus", and characterized by the formation of tumours with suppurating foci, usually in the tongue, face, jaw, or skin and subcutaneous tissue about the head and neck.

Horses are extremely rarely affected.

**Bacteriology and infection.**—The micro-organism responsible for the disease is the Streptothrix bovis or actinomyces bovis, a bacterium which has the power of branching in its growth. It is pleomorphic, existing in colonies, in which it grows in three forms, *viz.*, filaments, cocci, and clubs. If a drop of pus or scraping from a lesion is spread on a slide and examined, small granules visible to the naked eye are observed. If these are crushed under a cover glass and examined under a low power of the microscope, they will be found to consist of a number of club-shaped bodies arranged in the form of a ray with the thick end of the club outwards. This form is considered to be a late stage of development of the organism.

If a scraping or section from a growth at a younger stage is examined under staining, a network of very fine filaments is observed. These filaments constitute the active fungus. From the network branches shoot out, their growth appears to become arrested at their distal extremity, the sheath or covering of the filament at that part undergoes a mucilaginous degeneration, and clubs are thus produced; at the same time the protoplasm of the filaments in the network breaks up into coccoid bodies.

For ordinary diagnostic purposes staining is not necessary; colonies are easily detected under a low power; but for study of the parasite in its different forms, especially in sections, staining by carbolfuchsin (Ziehl Neilson Solution) and picric acid is recommended, the fungus staining red and the tissue yellow.

The disease generally occurs in isolated cases, and is not contagious in the usual acceptation of the term. When several animals are affected, it is usually from a common source, and not from one animal to another.

It appears to be more prevalent in swampy districts. The earlier view that the causal agent of actinomycosis is a saprophyte which gains access to the body from without, chiefly through barley, has been discredited since it became known that the actinomycotic organisms frequently present in the air passages of cereal plants are non-pathogenic, and consequently cannot originate actinomycosis.

**Symptoms and Diagnosis.**—These depend on the seat of inoculation. If the tongue is affected, raised nodules, which are firm and hard to the touch, are at first formed. Extending inwards from these there is marked proliferation of fibrous connective tissue, an indurated glossitis being set up, the tongue protruding from the mouth, and becoming as hard as a board, hence the term "wooden tongue". There is frequently

ulceration of the mucous membrane, but rarely suppuration. Prehension is interfered with, the animal becomes thin, and is in danger of starvation. This is the common seat of the disease.

Another seat is the outside of the face and the lower jaw, particularly the latter ("lumpy jaw"). Here a rarefying ostitis is set up, spongy cavities are formed, the skin ulcerates, and there is a discharge of the characteristic gritty pus. Sometimes the alveoli are affected.

The pharnyx is not infrequently affected, the growths forming polypi.

The skin and subcutaneous tissue, particularly in the region of the head and neck, are frequent seats of infection. In this situation it commences as a small tumour or "wen", increases in size to twice the size of a fist or more, sometimes being pedunculated, at other times broadly attached, sometimes also ulcerating. Lesions in this situation have been termed "clyers" or "loçus".

Almost all parts and organs can be affected, even lungs and liver, but the above are the principal seats.

**Differential diagnosis.**—It has been confused with osteosarcoma, carcinoma, and tuberculosis in cattle, and with botriomycosis of the horse, but the presence of the "ray fungus", the gritty pus, and the colonies, as seen by the naked eye when smeared on a glass slide, serve to distinguish it at once. It should also be remembered that actinomycosis is rare in the horse, while botriomycosis is comparatively common.

### How to deal with a case.

1. The disease is not a very fatal one, and cases are amenable to treament. No particular isolation is necessary, but pus from abscesses, soiled articles of dressing, or anything contaminated with discharge should invariably be destroyed. Instruments used for excision of tumours should be thoroughly sterilized by boiling. The coccus-like bodies or spores are fairly resistant.

2. **Treatment** is either medical or surgical, or both. Wherever possible excision should be practised, followed by disinfectant dressings, of which iodised phenol or Lugol's solution are recommended. Potassium iodide is a specific for the disease, and the best results are obtained from its administration in doses of 2 to 3 drachms daily, care being taken to stop it if symptoms of iodism show themselves. In three or four weeks a cure is effected, the enlargement of the tongue or tumour shrinking and disappearing. Biniodide of mercury may be substituted for the potassium iodide, given in 3 or 4 grains doses daily, dissolved with the assistance of ten grains of potassium iodide.

Where the tongue is affected, fluids should be given as food, and care exercised in the giving of medicines on account of liability to choking.

By administration of potassium iodide the milk supply is considerably reduced, and the iodine is largely excreted by the mammary gland, so during treatment the milk is not fit for consumption.

The flesh, when diseased parts are removed, is fit for food.

# NOTES.

## ANTHRAX.

**Synonyms.**—Splenic fever, splenic apoplexy, malignant pustule or malignant carbuncle, woolsorters disease (in human beings).

**Nature of Disease.**—Anthrax is a rapidly fatal disease of the blood due to the presence of the Bacillus Anthracis which increases with amazing rapidity causing destruction of the blood and loss of its function.

**Susceptibility.**—All animals may contract the disease. Young animals are the most susceptible. Flesh eating animals, including dogs, cats, pigs and the wild carnivora are comparatively little susceptible, though they may contract the disease by eating anthrax flesh. Habitual flesh diet may, however, confer immunity to a certain extent. Birds are immune, which fact may be noted in relation to the disposal of carcases by vultures, a process not recommended in the case of anthrax owing to the danger of development of spores. Frogs are also immune, an important fact in relation to water and soil infection. Birds and frogs are immune probably on account of the unsuitability of their blood temperature for growth of the organism. In the domestic animals, sheep (excepting Algerian sheep which are immune), cattle, camels and horses are susceptible in the order named. Man usually contracts the disease by inoculation (malignant pustule) or by inhalation in wool and hair factories (wool-sorter's disease). The wild herbivora, goat, deer, etc., are highly susceptible.

**Prevalence.**—The disease is not so prevalent in India as formerly; grass farms, with their clean grass-supply, having tolled its death-knell so far as cantonment animals are concerned. In former times when grass was brought in by grass-cutters, collected perhaps on roadsides and washed in dirty pools of water, it was much more common.

**Bacteriology and Infection.**—The Bacillus Anthracis is one of the largest of the pathogenic bacteria, and is readily detected by the microscope. To demonstrate it in the blood, clip the hair from the tip of the ear, prick lightly with a needle obtaining a small drop of blood, touch the drop with a cover glass and put on a slide gently squeezing the cover glass on the slide to spread out the drop of blood into a thin layer. Then examine under microscope using first the ⅙ inch objective, afterwards the 1/12 oil immersion: or touch the drop with a slide, draw another slide across the blood to make a thin smear, allow to dry in air and fix gently over spirit lamp, stain with a 1 per cent. aqueous solution of methylene blue for three minutes, wash off excess of stain with tap water, dry, mount with a little canada balsam and cover glass. The bacillus will be seen between the cells, singly, or in short chains. It is a straight rod, with characteristic square or slightly cupped ends, motionless, length about the diameter of a red blood corpuscle (length 5 to 6$\mu$ breadth 1 to 1·5$\mu$). The bacilli may be difficult to discover in the early stages of the disease, but in advanced cases they are found in great numbers in the blood and in the lesions, *e.g.*, enlarged spleen after death. As putrefaction soon destroys them, the blood of carcases should be examined as early as possible.

When confirmation of diagnosis is necessary, and if no microscope and accessories are at hand, smears of blood could be taken on two pieces of ordinary glass face together, and despatched wrapped in cotton wool to any institution where it is intended to have the blood examined.

The Bacillus Anthracis is a spore producing organism, and it is owing to spores that outbreaks continually crop up. Sporulation never occurs in the living animal body, but on exposure of the blood or body fluids to the air, the bacilli, which are aerobic, develop spores in a few hours. The greatest care should therefore be exercised in the spilling of blood, and *post mortems*, when necessary, should only be conducted at the place of incineration. The bacilli are easily killed, but the spore is very resistant, and ground once infected will remain so for many years. Outbreaks in India are invariably associated with recent rainfall. Under its influence spores germinate, grass becomes contaminated, and the infection in land is indefinitely kept up and disseminated.

Infection is usually produced by ingestion either through water or food. Grass is the chief medium. When cases are under treatment, and isolation measures are not sufficiently complete, infection can be conveyed by means of flies and biting insects. Infection by inhalation, as in woolsorter's disease in man, does not occur in the lower animals.

**Symptoms and Diagnosis.**—Anthrax manifests itself in different ways, depending on the seat of invasion, the amount of virus taken, susceptibility or otherwise of the animal affected, and whether the case is at the beginning or end of an outbreak, the later cases being usually milder.

An outbreak generally begins by one or several animals being found dead, usually animals in the best of condition. There may be blood or amber coloured serosity at the nostrils, eversion of the rectum showing dark coloured mucous membrane, rapid and great distention of the carcase and like conditions suggesting Anthrax, but the carcases of animals which die very suddenly may appear normal externally in all respects, therefore it is only by examination of the blood that the cause of death is determined. This form is termed **Fulminant or apoplectic anthrax,** and it is more usual in sheep and cattle than in other animals. If it is observed at all during life, there is dyspnoea, cyanosis, plaintive cries, convulsions, and death in from a few minutes to four hours.

Another type is **Anthrax Fever, Splenic Fever or Internal Anthrax.** This varies from an acute form ending in death within twenty-four hours to a subacute form with death after several days, or recovery. Varying with the intensity of the case we find very high fever, temperature 104° to 107° or even higher, dark congested visible mucous membrances amounting in some cases to cyanosis, sero-sanguineous or sanguineous discharge from nostrils, blood in the fæces which may be fluid, blood in the urine, excitement, sometimes very pronounced, a small accelerated, almost imperceptible pulse, great difficulty in breathing, tumultuous heart's action and emission of blood from the nose where there is great pulmonary congestion, severe colicky pains and tympany from intestinal complications (intestinal form).

In this internal form of anthrax, infection is contracted by ingestion. Bacilli or spores—in reality the spores, as the bacilli are killed by the gastric juice—are taken in by food or water, begin immediately to develop, and produce noticeable symptoms of the disease in three or four days on an average.

Cattle invariably suffer from this form of the disease. It is also commonly seen in horses.

The acute form is usually apparent at the beginning of an outbreak, the sub-acute type later on. The mortality from this form is from 70 to 90 per cent.

A third type is **External Anthrax,** a less severe form, and from which a good percentage of recoveries occur. It is a fairly common form amongst horses in India. It manifests itself by large diffuse swellings under the skin at the head, throat, neck, breast, shoulder, and other parts. These swellings appear suddenly, increase rapidly, and are hot and painful at first, but afterwards cold and doughy. There is no crepitation of the swellings as in Quarter-ill and no suppuration. Fever is not so high as in the internal forms of the disease, but symptoms described under Anthrax Fever may more or less be present. The durations of this form is from 3 to 7 days.

Infection is the same as in other manifestations of anthrax, but with the difference that in some cases during the prevalence of an outbreak, particularly those where the swellings are circumscribed, at the girth, on the ribs, in the flank, on the sites of old sore backs, scars, etc., or any vulnerable place, infection has been distinctly traced to the agency of flies, biting insects, saddlery, clothing, grooming kit, or soil, a local inoculation of the bacillus.

**Confirmation of Diagnosis.**—Diagnosis is not complete without either an examination of the blood, exudates of swellings, etc., by means of the microscope, when the characteristic bacilli will be found, or by *post-mortem* examination. The *post-mortem* appearances are characteristic. Speaking generally, they comprise—

(1) A black tarry condition of the blood, incoagulable or imperfectly so, and a shiny appearance.

(2) Enlargement of the spleen, commonly to two or three times its normal size but sometimes enormously. In very acute or fulminant cases there may be no enlargement of spleen, and in the horse enlargement is rare, but bacilli are found in it in enormous numbers.

(3) Yellow gelatinous exudate and hæmorrhagic patches in the swellings of external anthrax.

(4) Congested dark appearance of mucous membrane of intestines in the intestinal form, hæmorrhage into bowels, engorged mesenteric blood vessels, yellow exudate around lymph glands of mesentery, quantity of serous fluid in abdominal cavity.

(5) Dark colour of muscles and tissues excepting when there is enormous engorgement of the spleen and internal organs.

(6) Great distention of the carcase, little or no rigor mortis; early putrefaction.

(7) Blood or yellow exudate streaked with blood from nostrils; rectum everted, purple coloured and with bloody discharge from anus.

Diagnosis may also be confirmed by inoculation of a rabbit or guinea pig with blood. Both species of animal are susceptible to the bacillus and die quickly. The presence of the organism is determined by the microscope.

### How to deal with an outbreak.

Every outbreak must be dealt with on its merits after due enquiry as to the probable cause.

The main principles to be observed are :—

I. Evacuation of stables or lines.
II. Disposal of the subject whether dead or living.
III. Change grass or fodder supply.
IV. Disinfection.

1. Presuming that the outbreak has begun in the usual way by finding an animal dead in the stable or lines, or a case presenting symptoms in virulent form, **evacuate the particular portion of the stable** or lines in which the case has occurred, sending the animals of the sub-section, section, or troop into a picket a short distance from the stable or lines, and placing the immediate incontacts, *i.e.*, the animals on either side of the affected case, by themselves in isolation as suspects.

This sufficiently removes danger from the one case that has occurred, and admits of proper disinfection of the affected stable or lines which is so highly necessary in the disease.

2. Should there be more than one case and the outbreak more general, remove all affected squadrons, or the whole unit, as the case may be, into an isolation picket at least 400 yards away, placing all immediate incontacts in a picket by themselves.

Move picket after every case. It is essential to get away from ground soiled by infection.

3. **Disposal of carcase.**—The greatest care must be exercised in this. Remember that exposure of the blood, etc., causes the development of spores which are difficult to kill. Aim at the prevention of spore development in every way.

In removing the carcase from the stable or lines to the place of incineration or burial, be most careful to stop all natural orifices, using tow soaked in some disinfectant for this. also remove the animal's nosebag, clothing and line gear. Tie the head in such a manner, that drip from the nostrils will be prevented.

Never by any chance allow an Anthrax carcase to be dragged along a road. Use a cart or ambulance and thoroughly disinfect afterwards.

Completely incinerate the carcase in every case possible. If this cannot be done, bury **deeply** without opening or cutting of the carcase. The sides and bottom of the grave, and the carcase, should

be liberally sprinkled with quicklime, and the covering of the earth should be at least five feet.

Do not conduct *post-mortems* excepting at the place of incineration. Burn the soiled earth as well as the carcase, and be most careful to disinfect thoroughly the instruments, the hands and clothing of the person opening the carcase. Boil the instruments.

4. **Disposal of affected animals.**—Whether they should be destroyed or not depends on the nature of the case or outbreak. Confirmed cases without hope of recovery should be destroyed with the least possible delay. Cases showing a chance of recovery should be kept for treatment. In all instances where a limitation of the outbreak can be effected by early destruction, it should be carried out (paragraph 846, Regulations for the Army in India Instructions).

Affected animals should be destroyed in their standings or isolation picket to avoid the danger of scattering infectious discharges in transit. If they are moved alive to a place of destruction, incineration or burial, it should only be in an ambulance, under proper precautions, the ambulance being disinfected afterwards.

Destruction without spilling blood, which it is extremely necessary to avoid, can be easily effected by the injection of a saturated solution of strychnine into the jugular vein. Death is instantaneous. Be careful to disinfect the hypodermic syringe afterwards by boiling.

For the treatment of favourable cases, establish a Lazarette in some shaded convenient place, well away from unit-lines or picket. The utmost precautions must be taken in removal on account of the fear of infecting ground in transit. For this purpose the animal must have its nosebag put on, staling avoided by keeping it on the move, and dung gathered up and burned. On no account must animals be treated in the permanent standings of a unit.

5. **Change grass supply.**—This is a first essential on account of grass being the usual medium of infection. Outbreaks generally occur when grass is being fed green and brought in from certain districts. Make strict enquiry into these districts and the grass supply for the past few days equal to the usual periods of incubation of the disease. Change these districts, and sun dry all grass rations for three days. Destroy doubtful forage or bedding.

6. **Carefully take temperatures of all incontacts daily or twice daily** if numbers are not too great.

In outbreaks of Anthrax the term "incontacts" should not only include those animals standing on either side of an affected animal, but should be extended to all animals exposed to the original source of infection, *i.e.*, the grass supply, or whatever, from strict enquiry, is the assigned cause.

The greatest possible use should be made of the thermometer, as cases can be suspected or diagnosed in early stages thereby, and early removal is of great advantage in every way.

In this connection a knowledge of the "period of incubation" of the disease is necessary. This varies with the species of animal attacked, individual susceptibility, and the amount of virus taken in.

For general practical working purposes in India the following times may be taken as a guide:—

Horses and cattle 3 to 6 days.

Sheep 2 to 4 days.

Rabbits and guinea pigs on inoculation 24 to 48 hours, or perhaps a little longer.

7. *Disinfection.*—See "Routine of disinfection" under heading "General measures for dealing with outbreaks of Contagious Diseases". Remember the danger of spore development ; use fire and boiling water as much as possible and repeat disinfection process three times at least at intervals of three days. Particularly direct attention to forage, bedding, fæces, mangers, standings, clothing, nosebags, head collars, heel ropes, grooming and line utensils, saddlery and everything likely to have been contaminated by discharges from nose or mouth, by fæces and urine.

No half measures should be adopted, and if possible the disinfection should be under the personal supervision of a Veterinary Officer.

8. **Immunisation.**—Anthrax anti-serum is obtainable from Mukteswar Laboratory. *serum alone* confers immunity for so very short a time that as a preventive it is doubtful whether it is of any practical good. It is, however, useful as a curative agent, and may be injected intravenously in quantities of not less than 100 c.c. in animals suffering from the disease.

Immunisation with a spore vaccine prepared in the Insein Laboratory, Rangoon, has been carried out with good results during the last few years in districts in India where Anthrax is prevalent. More recently still a vaccine prepared by the Imperial Veterinary Research Institute, Mukteswar, has been used.

Method of use.—

(*a*) Thorough sterilization of instruments by boiling.

(*b*) Shake vigorously the bottle containing the vaccine both before and during use.

(*c*) Inject 1 c.c. of vaccine subcutaneously into the side of the neck. After injection massage gently to diffuse the vaccine.

No immunity is conferred until fourteen days after the inoculation, but thereafter a solid immunity lasting for approximately a year seems to be set up.

9. **Treatment.**—In India it is found that many cases, particularly towards the end of an outbreak, are amenable to treatment. Treatment should be antiseptic, and experience places Carbolic acid as the best remedy. Give three times daily in one drachm doses mixed with half an ounce of common salt, and administer in thick gruel and occasionally in linseed oil. If occasion requires, inject 5 to 10 per

cent. Carbolic acid solution into the carbuncles or swellings, and also apply the solution to the swellings externally. Treatment can be varied with other antiseptics. Be careful to burn all discharges, excrement, etc., use Chloride of lime freely on the animals' standing; give nourishing diet of a fluid nature, gruels, linseed, tea, etc. The blood being very thick, plenty of fluid is indicated.

Above all keep away flies and biting insects. The best method is to use 'smoke fires' of damp litter or other suitable material. Animals soon become accustomed to it, and the eye irritation which it causes is transient. When smoke cannot be used the attendant should be provided with a makkimar and be instructed to kill them, and the light application of cheer pine oil to the skin of both patient and attendant will keep them off.

10. **Return to lines.**—Fourteen days after the occurrence of the last case the outbreak may be considered over, and the isolated troop, squadron, or unit returned to its lines. This time will allow of the necessary course of disinfection of the lines. Note period of incubation of the disease in connection with freedom.

Animals cured of the disease should not join their unit until one month after recovery. Their clothing and line appurtenances should be thoroughy disinfected before they rejoin.

11. **Work during isolation.**—This depends on the circumstances of the outbreak, but as a rule there should be no work during the time calculated for incubation. After that animals should be placed in "Working isolation" up to their return to lines.

**Note** *re* **Prevention of outbreaks.**—A record should be kept in District Offices of all anthrax-infected districts and lands, and officers of mounted units should be informed of their existence, so that they may be avoided for grass supply. Past history of the disease can usually locate these places. The halting places and neighbourhood of stables on old dak roads, immediate neighbourhood of certain villages, grounds that were used for burial and *post-mortem* in days gone by when outbreaks were more frequent, are known to carry infection after the lapse of years.

When units are marching through a district they should be informed of these infected lands.

NOTES

**Synonyms.**—Bacterial Necrosis.—Necro-bacillosis.

**Nature of disease.**—The organism responsible for this disease is the Bacillus Necrophhorus. It gives rise to a variety of conditions in different species of animals, *e.g.*, Diptheria of calves, dry gangrene of the udder in cows, necrotic foci in the livers of cattle and sheep, inflammatory disease of the digits in cattle and sheep (foul in the foot, foot-rot), necrotic dermatitis, etc.

The particular form of Bacillary Necrosis with which this chapter deals, and the one of most importance to the Army Veterinary Officer, especially on active service, is the last mentioned, *viz.*, Necrotic Dermatitis or Necrosis of the skin of the extremities.

It is an acute local inflammatory disease characterised by the formation of lesions on the extremities, more particularly around the coronets and in the hollow of the heels. The lesions take the form of sloughs of the skin and underlying tissue.

**Susceptibility.**—The disease is more often seen in horses and mules owing to the conditions which favour its development.

**Prevalence.**—The disease is not prevalent in India, although cases occur from time to time. The conditions which favour the onset of the disease are continued exposure of the extremities to wet, cold and mud. These conditions have a debilitating effect on the tissues, allowing the organism, which is a saprophyte in the soil, to gain entrance through any abrasion of the skin. Hence under active service conditions, when animals are picketed in the open and exposed to inclement weather, especially when forced to stand in liquid mud up to their fetlocks, the disease is apt to assume an enzootic form. It may appear under such circumstances to be contagious, but more probably, when a number of cases occur at the same time, it is due to their being exposed to a common set of conditions, rather than to direct infection from animal to animal. The disease was very prevalent in France and Flanders during the first phases of the Great War. The systematic building of hard standings for all animals behind the firing line did much to reduce the incidence of the disease.

**Bacteriology.**—The organism as found in pus and necrotic tissue appears as a bacillus and also as long filaments which give it the character of a streptothrix. It is best stained with Loeffler's methylene blue, carbol fuchsin or carbol thionin. It does not form spores. It is an obligatory anaerobe. Pure cultures can rarely be obtained direct from lesions as the organism is usually mixed with other pathogenic or saprophytic organisms.

**Symptoms.**—There may be pain, swelling, and a slight discharge of thin serous fluid at the affected part, which is usually somewhere on the coronet, or in the hollow of the heel. In cases of acute necrosis there may be systematic disturbance, *e.g.*, high temperature, rapid pulse, etc. There may be acute pain and lameness. In other cases these symptoms may pass unobserved. In many cases the skin

is found to be already necrotic on the first appearance of lameness. When animals have been standing for sometime in mud, the commencement of the lesions in a number of cases will have passed unobserved, owing to the skin of the coronets and heels having been continuously obscured by the mud. The occurrence of one or two bad cases leads to a careful examination of the remainder of the animals of the unit, usually with the discovery of a number of other cases in which there are necrotic patches on the coronets. The necrotic patch is usually circumscribed, ranging in size from a shilling to a five shilling piece. It undergoes separation from the living tissue, and if removed as soon as separation commences, a superficial wound discharging a very little foetid pus remains. If neglected, the necrotic process is liable to extend into the deeper structures of the part and set up serious complications with irreparable damage. In such cases metastatic lesions may occur in internal organs with fatal results.

**Treatment and Prevention.**—Remove the cause. Every effort should be made to get animals on to dry standings. Daily inspections should be made for wounds on the coronet or heel, and any such cases should be removed at once to a suitable place for treatment. On active service it might not be possible to move all the animals of a unit on to dry standings. In such circumstances it is all the more important that a regular daily parade should be held away from the muddy lines for the detection of wounds and commencing necrosis. It is always possible to find some sort of shelter and dry standings for the treatment of a few animals. Severe cases should be evacuated at once to hospital. Under peace conditions all cases should be admitted to hospital for treatment. General treatment consists in removing all necrotic tissue and treating the resulting wound with ordinary antiseptics. Cauterisation and curetting may be indicated in some cases. Poulticing is contra indicated. The wound should be covered with a pad and bandage. Careful attention to sanitation is all important. The disease should be regarded as contagious and reported as such. Cases under treatment should be properly isolated. Under peace conditions particular attention should be given to avoiding abrasions and cracked heels. Legs should be thoroughly dried on return from work in wet and muddy weather. Should a number of cases occur in a unit, all dressing of wounds should be done under careful supervision and at a central dressing station. The dressing of wounds in unit lines should be stopped. The blistering of horses during an outbreak is attended with risk as it pre-disposes to infection.

# NOTES.

# NOTES.

## BLACKQUARTER

**Synonyms.**—Blackleg. Quarter-Evil, Quarter-Ill, Strike.

**Nature of disease.**—An acute infectious and very fatal bacterial disease of the ox and sheep characterised by fever, emphysematous swellings and lameness. It is caused by the Bacillus or Clostridium Chauvoei, one of the group of organisms which produce gas gangrene and are facultative parasites. Another of the group, the bacillus of malignant oedema, produces symptoms very similar to those of Blackquarter but is pathogenic to the equidae as well as to cattle.

**Susceptibility.**—Affects cattle and sheep only. Cattle are most susceptible between the ages of 6 months and 2 years, but the disease may occur in young calves and adult cattle.

**Prevalence.**—The disease is prevalent all over the world.

**Infection.**—The natural method of infection is not known with certainty but the organisms probably enter by means of abrasions of the skin.

**Symptoms.**—Usually the first thing noticed is lameness which may be limited to one hind-leg, accompanied by blowing respiration, grunting and high temperature. A local swelling may be discovered, usually in the upper part of a hind-leg, but at other times on the shoulder, neck or abdominal wall. The swelling is at first hot and painful and rapidly extends, involving a large area. Later it becomes cold and painless. The swelling, on manipulation, is found to be emphysematous, crackling on manipulation. If cut into it is seen to be engorged with dark red bloody exudate, more or less frothy. Death rapidly ensues. The disease in sheep runs a much more rapid course than in cattle, the animals frequently being found dead. In this respect it resembles Anthrax. Local swellings are not usually so well marked as in cattle.

**Diagnosis.**—The characteristic lesion is the emphysematous swelling crackling on manipulation. If cut into, the muscles and tissues of the part are dark red or blackish, and have a porous and dry looking appearance, due to separation of the fibres by gas produced by the organism. In other parts there may be bloodstained exudate and hæmorrhage into the muscular tissue. The affected tissues have a characterisitic sour odour like rancid butter. The lymphatic glands of the part are swollen and often haemorrhagic. There may be blood-stained exudate into the pleural and peritoneal cavities.

**Treatment.**—Ordinary treatment by medicines is of no avail. Blackquarter has a peculiar distribution. There may be certain localities or fields which are known to be infective, whereas others in the immediate vicinity or actually adjoining may be clean. Hence preventive treatment includes the avoidance, as far as possible, by cattle of a susceptible age, of localities where the disease is known to occur. The ploughing up and treatment of infective pastures with lime is recommended. The carcases of animals that have died

of the disease should be carefully disposed of by cremation or burial, together with any escaped discharges or soiled earth.

**Immunisation.**—Various methods of vaccination against Blackquarter are practised, *viz.*, Arloing's Kitt's, and Leclainche and Vallee's methods, etc. The two former methods involve the use of vaccine prepared from the muscular tissue of fresh lesions and which contain the organism in an attenuated form. Leclainche and Vallee's method involves the use of an anti-serum followed by an attenuated pure culture. A serum simultaneous method is also recommended. The Imperial Veterinary Research Institute, Mukteswar, now issues "Blackquarter Aggressin" prepared from different strains of the Bacillus Chauvoei and therefore a polyvalent aggressin. It is claimed that this aggressin is safe as it contains no living germs, and is also efficacious and easily administered. It is recommended that animals be inoculated about one month before the onset of the season when outbreaks are known to occur. A second inoculation is recommended to be given 9 or 10 days after the first to strengthen the immunity. Immunity is believed to last for at least a year. This means that if animals are inoculated as calves or yearlings it will carry them over the most susceptible period of their life.

An anti-Blackquarter serum is also manufactured which is useful in cutting short an outbreak by inoculating all animals exposed to infection, but the protection only lasts for about 9 days.

NOTES.

## BOTRIOMYCOSIS

*Synonym—Discomycosis.*

**Nature.**—This is a disease principally affecting horses, due to the inoculation of a micro-organism formerly known as the botriomyces or discomyces but now considered identical with the Staphylococcus pyogenes aureus, and characterized by the formation of chronic inflammatory connective tissue growths or tumours with suppurating foci. A growth of this nature is termed a botriomycoma.

**Bacteriology and Infection.**—Two forms of the Botriomyces equi are found in lesions, *viz.*, the isolated or discreet micrococci and diplococci, and secondly aggregations of cocci (Zooglea).

**Aggregations.**—These are generally found in chronic lesions. The smears made of Botriomycotic pus from a lesion more than a week old show minute granules which can be seen with the naked eye. Under a low magnification these granules are observed to be circular and the surface bossellated. This appearance is due to the fact that each granule is composed of a number of smaller spherical masses. If the granule is crushed, stained and examined it is observed that the mass is composed of micrococci which appear to be embedded in an amorphous material, and it is suggested that the masses are enclosed in a thin envelope or membrane. In a stained section of a botriomycotic lesion it is found that the micrococci in the centre of each mass have lost the power of retaining stains. It is suggested that the colony formation is a process connected with increased resistance of the organism, or a process of retrogression or degeneration. The so called Botriomyces equi is readily stained with the usual stains, and is indistinguishable from the Staphylococcus Aureus.

The disease is produced by wound infection, and the wound may be insignificant such as a scratch. The usual causes are wounds from some part of the harness or following castration.

It generally occurs in single cases, and has no tendency to spread to other animals in a stable. Pus, all the same, is infective. Its development is slow and insidious, so much so, that horses castrated as yearlings and then infected, may not shew the disease until they are five or six years old or even longer.

**Symptoms and diagnosis.**—As in actinomycosis symptoms are limited to the local lesions. Tumours may be single or multiple, and vary in size. They may be the size of a pea, or as large as a man's head. Their common seats are the front of the shoulder (shoulder tumour), spermatic cord (producing a schirrous cord) the point of the elbow (elbow tumour or shoe ball) and the udder. The main substance of the tumour is dense fibrous tissue. A varying number of abscesses are contained in it. The skin over the enlargement may point and an abscess bursts discharging a thick brown-pus. The wound thus produced heals up, and other abscesses form, repeating the process. The pus is characteristic, being sticky and containing granules, but unlike those of actinomycosis they are not gritty when rubbed between the finger and thumb.

## How to deal with the disease.

1. Excise the tumour whenever possible. It is necessary to practise ligation freely as tumours are usually very vascular.

2. Administer potassium iodide internally. It is considered almost a specific, but its action is perhaps not so marked as in actinomycosis. It may be of value in instances where the growth is so diffuse as to render operation impossible, or it may reduce the growth to an operable condition.

3. As a wound dressing a solution of iodine, ½ to 1 per cent., is recommended.

4. Soiled surgical and wound dressing should be burned: instruments should be thoroughly disinfected after use.

## Bursatti.

Bursatti is the name used in India and the Soudan to designate a particular diseased condition of the skin and subcutaneous tissues of equines characterised by the formation of fibrous tumours, embedded in which are peculiar bodies known as "Kunkurs", and ulceration of the overlying skin.

The disease has a seasonal distribution, the majority of cases occuring during the rainy season (June to October), as indicated by the name.

**Symptoms.**—The first evidence of the disease is the development of a hot painful swelling in the subcutaneous or submucous tissue, the overlying skin or mucous membrane remaining intact.

Within about a week the swelling becomes harder followed by thickening and adherence of the skin to the underlying tissues.

In about a fortnight or three weeks later the skin covering the lesions sloughs, leaving an open sore, and margins of which are thickened and fairly regular.

Yellowish red granulations develop over the ulcerated surface which becomes raised above the level of the skin and embedded in the ulcerated surface are a number of small rounded or irregular bodies, which can be enuceleated by pressure, leaving small cavities.

These bodies, known as kunkurs, vary in size from a pin's head to a pea. In the early stages of the disease they are of a dense fibrous consistency, later they may undergo partial calcification.

Lesions not exposed to friction or injury show little tendency to spread, but those situated in places exposed to irritation of any kind often attain a very large size.

The lesions frequently develop in connection with wounds. The parts most frequently involved are the lips, eyelids, and especially the region below the eye at the inner canthus, nasal alæ, neck, withers, shoulders, pasterns and fetlocks, sheath and penis.

The affected regions are the seat of excessive itching. There is practically no pus formation in uncontaminated lesions.

**Histology.**—Microscopical examination of sections shows that the tumours are composed almost exclusively of fibrous tissue.

**Etiology.**—Various views have been put forward regarding the nature of the specific cause of the disease, *viz.* :—

1. That it is due to filarial embryos (Rivolta).
2. That it is caused by a fungus (Holmes).
3. That it is identical with summer sores, due to the larvæ of Habronema deposited in the skin by the invertebrate hosts (*i.e.*, the common horse fly for H. muscæ and H. megastoma, and Stomoxys irritans for H. miscrotoma).

The actual cause of the disease is still undetermined.

**Treatment.**—1. Extirpation of the fibrous tumours and dressing of the wound with red oxide of mercury.

2. Curetting and the application of powdered potassium permanganate.

3. Red iodide of mercury 5 to 20 grains with potassium iodide 1 to 3 drachms given daily in the feed or drinking water.

4. Van Saceghem's specific for summer sores—composed of the following :—

| | Parts. |
|---|---|
| Plaster of Paris | 100 |
| Alum | 20 |
| Napthalene | 10 |
| Quinine | 10 |

Applied after thorough cleansing and disinfection of the sore. The dressing dries the sore rapidly and prevents the attacks of flies.

5. Quinine sulphate used as a dressing has given good results.

Removal of badly affected animals to another part of the country has been recommended.

Treatment with spleen extract injected intra-muscularly and intravenously in increasing doses has been tried with varying success. In some cases healing occurred with great rapidity, in others the treatment had no beneficial effect.

Neoarsphenamina, B. P. internally, and local treatment with Ox-bile have also been tried with varying results.

Although there is no evidence at present to indicate that the disease is transmitted from animal to animal by any outside agency it is advisable to isolate affected animals.

# NOTES.

**Nature of disease.**—Coccidiosis is a disease affecting for the most part the intestinal tract and its appendages.

Depending on the species of animal affected it may give rise to acute and rapidly fatal disease characterised by symptoms of diarrhœa and dysentery, or to a milder form of disease, in which parasites may be harboured indefinitely without the exhibition of any marked clinical symptoms.

The casual organism is a protozoan parasite, the coccidium. Parasites of this type are found in many-vertebrate and invertebrate animals and a large number of species have been described.

**Susceptibility.**—Rabbits are most commonly affected and among them the disease may give rise to serious losses. It has also of late years been found to be widespread amongst cattle and may cause serious losses among young animals. Losses amongst sheep and goats have also been reported from the same cause. Poultry and game birds may also be seriously affected. Coccidia have been found in dogs and cats but the disease in these species is relatively unimportant.

**Prevalence.**—The question of the existence of Coccidiosis in India has been the subject of careful study at the Imperial Veterinary Research Institute, Mukteswar, for several years past and according to Edwards it is now fairly well established that cattle, sheep and goats are probably ubiquitously carriers of coccidia in India and seldom show any ill effects as the results of latent infection. Enzootic outbreaks and sporadic cases may occur. He states that the most serious form of coccidiosis in cattle is that which occurs as a sequel to rinderpest; that in the course of the specific depression in resistance that takes place in the intestinal epithelium in the course of this disease the restraint upon the multiplication of the dormant coccidia is relaxed and the resuscitated parasites frequently bring about secondarily a fatal termination in what would otherwise be a mild or a sub-acute attack of rinderpest from which the animal would recover. With regard to the distribution of the disease in India, Hugh Cooper remarks as follows :—

"This disease appears to have a peculiar distribution in that it has been reported most frequently in mountainous or hilly regions. The reason for this is probably the existence of moisture conditions in these regions necessary for the development of the oocysts in the outside world. Valleys or flat country, either allow an excess of water to remain or else are exposed to greater dessication, and in either event they are unsuitable for the development of oocysts."

It was in the mountainous regions of Switzerland that bovine coccidiosis was first discovered in 1878.

**Protozoology and Infection.**—The casual parasite, *i.e.*, the coccidium has a rather complex life cycle. As found in the fæces of rabbits examined under a low power of the microscope it is a small ovoid body like an immature bird's egg. Rounded forms may also be found, and the size varies from 22—50 μ long by 13—28 μ broad. In cattle the coccidium varies more in form and dimensions and the rounded form is the com-

moner. The dimensions of the ovoid form in cattle are 20—25 μ by 10—12 μ and of the rounded form μ 12—15 .

The parasite has a double contoured envelope. One pole is narrower than the other and is slightly flattened. At this point there appears to be an orifice in the capsule (micropyle). The protoplasm may completely fill the capsule and is coarse and granular ; but in the majority of parasites it has undergone considerable shrinking and is in the form of a coarsely granular ball like mass in the centre of the Capsule, in which a nucleus can sometimes be seen with difficulty. The remaining space is occupied by transparent liquid. These parasites as seen in the fæces are termed oocysts. Further development of these oocysts takes place outside the animal body under favourable conditions. When ripe they are capable of infecting other animals of the species for which they are specific if ingested, and then undergo further development.

The life cycle of a coccidium is rather complex. In cattle they invade the epithelial cells of the mucous membrane of the intestine, more particularly those lining the villi and crypts of Lieberkuhn. They may also be found in these situations in rabbits, but in this species the commonest site of lesions is the liver where the parasites are found distending bile-ducts. There is an asexual method of multiplication within the epithelial cell, followed by a differentiation into male and female elements and a sexual method of multiplication. The fertilized female (oöcyst) gains access to the outer world and becomes divided after an interval into a number of daugher elements (spores and sporozoites) enclosed in a resistant capsule. This forms the egg body or ripe oocyst. This oocyst when ingested by a healthy susceptible animal gives rise again, after liberation of its sporozoites by a process of sex-multiplication within the epithelial cells of the host, to an enormous number of parasites (merozoites) and thus disease is produced and the cycle repeated.

The more important coccidia of the domesticated animals belong to the genus Eimeria.

Recent researches have established the specificity of different genera of coccidia for different animals. The disease Coccidiosis assumes most importance in the case of the rabbit. It will not be out of place therefore to commence with a brief description of the disease in this species of animal.

## Coccidiosis of the rabbit.

**Symptoms and Diagnosis.**—The casual parasite is now designated Eimeria steidœ.

Young animals are most acutely affected, and death may occur quite suddenly before oocysts are found in the fæces. The usual symptoms are dullness, loss of appetite, wasting, pale or jaundiced membranes, pot belly, tympanitis, diarrhœa and finally paralysis and death.

In older rabbits the disease may last for 2 or 3 months, and in other cases, although infected they may not show any symptoms, but harbour the parasites and act as carriers. Diagnosis is confirmed by an examination of the fæces and detection of the oocysts.

*Post-mortem* appearances are characteristic. The liver is usually enlarged, in advanced cases to a very considerable extent. On its surface are seen a number of white patches or spots, many of which are elongated in shape, and represent bile ducts distended with coccidia. If one of these spots is opened a milky fluid escapes. This fluid examined under the microscope is seen to contain enormous numbers of oocysts. The spots or patches vary in size from a millet seed up to a hazel nut. There may be no visible lesions in other organs, but in many cases lesions of the small intestine may be observed in the form of congestion and sometimes white points or spots which represent masses of coccidia situated in epithelial cells lining the villi and crypts of Lieberkuhn.

**How to deal with an outbreak.**—Medicinal treatment is of no avail. All infected rabbits should be destroyed. Having in view the fact that infection can only take place by the ingestion of food contaminated with fæces of infected animals and that for the ripening of voided oocysts moisture and oxygen are necessary, frequent moving (every 3 days) of apparently healthy rabbits to clean hutches or runs is recommended. Young rabbits should not be placed in the same hutch or run with older rabbits, which may be carriers of the disease. The most thorough routine disinfection of soiled hutches and runs should be carried out.

**Coccidiosis of cattle.**—The casual coccidium is named Eimeria Zurni.

Reference has already been made to the prevalence of this disease in India. Bovine coccidiosis would appear to have a world-wide distribution and has been reported in most European countries, in North-East and South Africa, America and Australia. In European countries the disease occurs far more frequently at pasture. Wet years and damp marshy pastures favour the incidence of the disease. It is essentially a disease affecting young animals from 6 months to 2 years old. Outbreaks have also been reported in very young animals 4 to 7 weeks old, but older animals are rarely affected.

**Symptoms and Diagnosis.**—Usually the first symptom noticed is diarrhœa, but the expulsion of blood clots with normal fæces may precede diarrhœa. There may be straining and evidences of pain. As the disease progresses the fæces become mucoid in character and large quantities of blood may be expelled. Concurrent symptoms are dullness, loss of appetite, wasting, and irregular rumination. Later, the fæces become very liquid and offensive and the blood clots become still larger. The temperature is very little raised although the pulse and respirations are accelerated. The patient becomes very weak and after about 10 days, either takes a turn for the better when recovery may take place, or the disease progresses rapidly to a fatal termination. The disease may assume a sub-acute or chronic form. The period of convalescence may last up to several months—one attack is said to produce a strong immunity, but the parasite may remain in the alimentary tract and give rise to a subsequent breakdown, if the animal's constitution is weakened by any other disease.

The clinical picture is usually sufficient to enable one to make a diagnosis, which may be confirmed by a microscopical examination of the fæces. Coccidia are usually found in abundance in the shreds of mucous covering the fæces. Fæces can also be examined for coccidia by "Sheatner's sugar flotation" method.

**Prognosis.**—In some European countries the disease causes serious losses and the death rate may be as high as 10 to 15 per cent. Older animals stand a much better chance of recovery.

**Differential diagnosis.**—In India the disease as previously mentioned is not of itself of great economic importance, a large percentage of cattle being constant carriers of coccidia. When, however, affected animals become infected with other diseases, such as Rinderpest, Johne's Disease or Piroplasmosis, the combination may cause serious losses which would otherwise not have occurred.

*Post-mortem* examination of animals that have died of coccidiosis reveals lesions which vary according to the length of time the disease has been in existence. The lesions may be confined to the rectum, the mucous membrane of which is thickened and congested, and the epithelium, detached at many points, so as to present a wrinkled surface dotted over with hæmorrhagic points and often covered with blood stained mucous. The mucous membrane of the Colon and Cæcum may be congested and thickened, in which case the bowel contents may be the consistence of clear soup mixed with shreds or flakes of clotted blood. In cases lasting 8 to 10 days the intestine is affected throughout; although the rectum still shews the most marked changes. The mesenteric glands are enlarged, the mucous membrane of the large intestine is wrinkled and covered with brownish grey patches and shallow ulcers where the epithelium has been shed. The contents of the bowel are liquid, offensive and small in quantity. There is extreme emaciation and the muscles are macerated in appearance. The main histological changes are a destruction of the epithelium of the mucous membrane of the intestine and of many of the crypts of Lieberkuhn. Coccidia are found abundantly in the fæces which are mixed with blood and mucous. They are also found in the cells and free in the lumen of the crypts of Lieberkuhn. In the last stages the changes in the rectum are deeper seated and there are large cavities extending down to the submucous coat.

With reference to Coccidiosis of cattle in India, Hugh Cooper remarks that the most striking feature of *post-mortem* examination of an animal that has died of coccidiosis is the frequent absence of naked eye lesions, in spite of, perhaps, a history of severe intestinal disturbance. Infection by coccidia is almost entirely limited to the mucous-membrane of the large intestine, and in spite of the fact that large quantities of blood may have been passed in the fæces by the animal during life, very little alteration in the intestine to account for this may be seen. This is due to the fact that escape of blood takes place from a large number of very minute ulcers, which may be even microscopic in size, or only just appreciable to the naked eye as very small, apparently insignificant blood spots upon the lining of the intestinal wall.

### How to deal with an outbreak.

**Preventive measures.**—The same principles apply as in the case of coccidiosis in the rabbit. In countries where the disease is enzootic and causes serious losses from time to time, the following measures are to be recommended.

Young cattle should be removed from damp pastures or land known to be infectious. Affected animals should be strictly segregated, and young cattle should not be allowed to run with adult cattle which may be carriers of the disease although shewing no clinical symptoms. Great importance is attached to keeping young cattle away from marshy and boggy pastures. In the case of cattle kept in paddocks, enclosures or byres, it is very important to see that the ground is kept sweet and dry, and that excreta which is the main source of infection is properly disposed of by burning or other means.

**Treatment.**—Various forms of treatment have been recommended. Astringents and antispasmodics have been used with a success, *e.g.*, catechu, Asafœtida, Tr. Opii, etc. Thymol has also been strongly recommended. Diarrhœa should be combated by opiates, astringents and demulcents. During convalescence tonics and good feeding are indicated.

With regard to the disease in India Hugh Cooper remarks as follows :—Methods devised to prevent the spread of coccidia have been recommended, but in view of the fact that latent infection already exists in probably nearly all cattle in India, these methods are of little practical value in this country. Unfortunately also, up to the present time, all types of coccidia have shewn themselves to be remarkably resistent to every form of curative treatment that has so far been tried.

### Coccidiosis of Sheep and Goats.

This disease has been reported as occurring in Great Britain, France and Northern Africa. So far as India is concerned Edwards states that sheep and goats are probably ubiquitous carriers of coccidia in India, and seldom show any ill effects as the result of the latent infection. The sheep, and goat each has its own specific coccidium.

The disease affects particularly young animals with symptoms resembling those seen in Coccidiosis of the rabbit and ox. Lesions are found in the form of innumerable white spots in the mucous membrane of the small and sometimes large intestine. The lesions sometimes take the form of papilliform outgrowths of the mucous membrane, each about the size of a pea. Numerous coccidia are found in the mucous membrane covering these outgrowths. There may be other areas of congestion of the mucous membrane. Adult sheep and goats may harbour coccidia in their fæces without showing manifest symptoms of disease.

Preventive treatment as recommended for rabbits may be tried.

# NOTES.

## CONTAGIOUS ABORTION OF CATTLE

**Synonyms.**—Epizootic abortion, Brucellosis.

**Nature of Disease.**—Contagious abortion is a specific catarrhal metritis, the act of abortion being merely a symptom of the disease.

Contagious abortion causes very serious losses in dairy herds. In some cases, when abortion takes place late in pregnancy, the calf may be born alive, but is invariably weak and usually dies. The losses however include not only the loss of calves, but a serious drop in the milk yield, since in many cases abortions occur in the earlier stages of pregnancy.

Infected cows may abort a second and even a third time, but provided that no fresh animals were introduced into an infected herd the disease would in time tend to work itself out; but this natural process would extend over some years.

Cows that have aborted may become temporarily sterile.

The disease is usually introduced into a herd by the purchase of a new and infected cow. In an outbreak the number of abortions are usually few in the first year, and increase during the second year, reaching their maximum during the third year.

**Susceptibility.**—The organism causing Contagious abortion of cattle would, in natural circumstances, appear to be specific for cattle. Other species of animals are liable to suffer from Contagious abortion, *e.g.*, mares and sheep, but the causal agent appears to be specific for each species.

**Prevalence.**—Contagious abortion of cattle has a world wide distribution. In India the disease has probably been in existence for many years, but it is only within recent years that it has received special attention, as the result of which it has been found to be very widespread, and has been demonstrated to exist in many of the dairy farms situated in different parts of the country.

**Bacteriology and Infection.**—The casual organism was first demonstrated by Bang in 1897 and is known as Bang's Bacillus or the Brucella abortus. It is essentially a tissue parasite, the predilection seat of which is the gravid uterus, in which it is found in large numbers, either free or contained in cells in the uterine excretion of aborting cows (Bang). The bacillus is a small non-motile and non-sporing cocco-bacillus. In lesions and in artificial media it frequently occurs in clumps, and in the latter is often polymorphic. It stains readily with the basic aniline dyes but is Gram negative. The larger bacilli frequently shew granular staining, resembling a number of small streptococci. There is some difficulty in obtaining primary cultures, but on sub-cultivation it grows readily under ordinary aerobic conditions. The common method of infection is by the ingestion of fodder contaminated with uterine discharge after abortion, particularly the foetal membranes which teem with the bacilli. It may be possible for the disease to be transferred by the bull, although he may shew no symptoms of infection. The disease can be conveyed experimentally to the mare, ewe, goat, and rabbit by ingestion or parenteral injection. From the results of recent work in England and

elsewhere it would appear possible that some cases of undulant fever in men may be due to infection from bovine sources.

**Symptoms.**—There may be no definite premonitory symptoms, but on the other hand there may be the usual signs of approaching labour. In cows, which are in milk there is some falling off in the milk yield, and the milk may resemble colostrum. There is an opaque vaginal discharge which may appear a short time before the act of abortion. Abortion usually takes place between the 3rd and 7th months. If the abortion takes place late in pregnancy the calf may be born alive but in such a case is usually weakly and frequently dies. Retention of the membranes is common after abortion, and cows that have aborted often shew abnormally frequent oestrum, difficulty in conception, and may become sterile.

**Lesions.**—The lesions are confined to the uterus, and the typical lesion can best be seen in an animal about to abort. Between the uterus and the chorion is a thick yellowish, glairy, odourless exudate, and there may be a gelatinous oedema of the foetal membranes which gives them a characteristic appearance after abortion. The cotyledons often appear as though macerated, opaque and of a dull greyish colour. The causal organism may be recovered in pure culture from the exudate and membranes, before or immediately after abortion, and from the liver, heart, blood, and alimentary canal of the aborted foetus and foetal cotyledons.

**Diagnosis.**—The disease is suspected when a number of animals abort at different times. The nature of the discharge which is glairy and yellowish will help to confirm one's suspicions. The chorion is thickened and oedematous and the cotyledons yellowish grey coloured and softened. It may be possible to demonstrate the Brucella abortus in smears from the placental exudate, or foetal liver, or intestines, but a negative result proves nothing. Often cases of contagious abortion occur without the exhibition of any characteristic symptoms, and the only means of confirming one's suspicions is by means of serological tests, *viz.*, the Agglutination Test and the Complement Fixation Test. A reaction to either of these tests indicates that a cow is or has been infected with the Brucella abortus, but it does not necessarily follow that the cow will abort.

**How to deal with an outbreak.**—A strict look out should be kept for any cows shewing premonitory symptoms of abortion, and any such should be immediately isolated. In the case of cows that abort in the herd, thorough measures of disinfection should be carried out with regard to the aborted foetus and any uterine discharges. The foetal membranes should be burned or buried in quick lime. Contaminated litter or soil should be removed and burned. The uterus of the affected cow should be irrigated with disinfectant solution, and the soiled thighs and legs carefully washed with a disinfectant. The floors and walls of the building should be thoroughly disinfected according to routine measures. Newly purchased animals should be tested by the Agglutination Test before being admitted to the herd. Cows should not be sent away to the bull. A bull which has served infected cows should have his sheath and penis thoroughly washed with disinfectant.

**Protective inoculation.**—It has already been stated that infected animals gradually acquire an active immunity, so that after the first abortion they may carry the foetus to the full time, although many will abort a second time and much more rarely a third time. Vaccination against the disease is practised. Reports on the results of vaccination in different countries vary, but it would appear that if systematically carried out on selected herds, in which too gross an infection has not had time to become established, favourable results may be expected and a considerable reduction in the number of abortions may be anticipated.

Two kinds of vaccine are employed, *viz.*, a vaccine containing dead organisms and a vaccine containing living but attenuated organisms. The sterilised vaccine may be used in the case of pregnant cows and is injected at monthly intervals up to the sixth month of pregnancy. The living vaccine is only used in the case of non-pregnant animals, and should be injected two months before the commencement of pregnancy.

Results in India from the use of sterilised vaccine have not been favourable and its use has been discarded in favour of the live vaccine method. There are a number of points to be considered when deciding upon the best measures to be adopted in controlling an outbreak of contagious abortion. The best results can only be expected from vaccination with living vaccine in herds in which the disease has only just commenced. If wholesale vaccination is attempted, it must be continued to include all new entrants to the herd. It is important to remember that just as an infected animal that has acquired immunity may remain a carrier for years, so also may an animal vaccinated with living vaccine remain a carrier. The bacilli lie latent in the udders of carriers and are excreted in the milk. With succeeding pregnancies the bacilli reinvade the chorionic tissues, but their effect is less severe with each succeeding pregnancy. Young animals until the time of sexual maturity appear to be immune.

In recent years experimental work with a third kind of vaccine has given very promising results in the U.S.A. and Great Britain. This kind of vaccine is prepared from smooth avirulent or low virulent abortus strains and is inoculated in the living state into non-pregnant animals. Trial vaccination of calves and heifers in selected Military Dairy Farms has been carried out but it is too early yet to judge the results of these trials.

In conclusion, when dealing with a fresh outbreak of contagious abortion in a herd in which it is decided to vaccinate, the first thing to do is to test all animals by means of the Agglutination Test, segregate infected animals, and vaccinate the healthy animals. The Agglutination Test should be repeated after one month and thence forward about twice a year.

Blood for agglutination test should not be taken less than 14 days after abortion.

## EQUINE CONTAGIOUS ABORTION.

**Nature of Disease.**—This disease is similar in nature to the disease in cattle, in that it is a specific metritis of which the act of abortion is a symptom. McFadyean and Edwards confirmed the findings of previous observers and established the fact that contagious abortion of mares is a specific disease due to the bacillus abortivo-equinus.

**Susceptibility.**—Donkeys are equally susceptible with horses.

**Prevalence.**—The disease has probably a world wide distribution. It is well established on the continent of Europe and the United States of America. In India investigations in recent years have proved the disease to exist in a number of horse breeding studs throughout the country. Amongst donkey mares in India it existed in one particular stud to the writer's knowledge 20 years ago, and at the present time is still in existence in this same stud.

**Bacteriology and Infection.**—The common causal organism of Contagious equine abortion is the bacillus abortivo-equinus, in India however many cases are due to other organisms normally not pathogenic, *e.g.*, B. Coli which gain entrance to the gravid uterus and produce a metritis resulting in abortion. In the case of contagious abortion, due to B. Abortivo-equinus the affected animal may be a carrier for 2 or 3 years, after which she may become clean, and then become reinfected again (Edwards). The fæces and possibly the urine also of a carrier may be infective. The commonest source of infection is fodder contaminated by the uterine discharges, placenta, or foetus of a mare which has aborted, and the commonest method of infection is by ingestion. The predominant form of the organism is a coccus or short plump bacillus measuring from 5 to 1 μ in length, and 3 to 5 μ in width. Distinct rod-shaped elements are usually also present. The organism has a marked tendency to pleomorphism, both in the animal body and in artificial cultures. It stains irregularly, with the basic anilin dyes, sometimes showing bipolar staining and is Gram negative. It grows upon the ordinary media under aerobic conditions. The growth upon agar is characteristic. After two or three days incubation the surface growth shows a peculiar wrinkled appearance which resembles tanned aligator's or lizard's skin. The peculiarity is only seen in rich cultures of the organism (McFadyean and Edwards). The organism is motile and does not form spores.

Abortion can be produced experimentally in other species, *e.g.*, ewes and cows, by the intravenous injection of cultures of the bacillus abortivo-equinus.

According to Edwards, the disease may spread and intensify over a number of years causing formidable losses. In other cases although testing shews a high percentage of infected animals, the abortion rate may be low. An example of this latter phenomenon is the disease as it has existed in a particular Remount Stud for some years past. Tests have revealed a high percentage of infection, whereas the actual abortions have been comparatively few. Edwards suggests that the probable explanation of the intensification of the disease is the conditions under

which brood mares are kept. When they are more or less congregated in a small area, there is the greater liability to heavy soil infection as a result of which animals are likely to ingest comparatively massive doses of infective material.

**Symptoms and Diagnosis.**—As in the case of contagious bovine abortion, the disease is a specific metritis. There is a chocolate coloured discharge from the vagina with a characteristic sour odour. This same odour may be noticeable in the foetus and membranes. Cases of abortion, however, may and do commonly occur, without any premonitory symptoms. There is nothing clinically diagnostic in a case of abortion to shew whether it is sporadic or contagious. If other cases have ocurred in the stud at different intervals, one's suspicions would be aroused and, the nature of the discharge might help in the diagnosis. Microscopical examination of smears from the exudate or membranes may reveal the causal organism: and cultures may be obtained from the same if not contaminated with other organisms. The most convenient and reliable methods of diagnosis are by the serological tests, *i.e.*, agglutination and complement fixation, of which the former is most commonly used.

**How to deal with an outbreak.**—Medicinal treatment is of no avail. Vaccines when the disease is caused by B. Abortivo-equinus have met with some success in some studs. In the Remount Studs in India however the most effective means of controlling the disease have proved to be sound hygienic and isolation measures. Foaling boxes so constructed that they can be easily and thoroughly disinfected are essential and the utmost cleanliness during and after parturition.

A wide dispersion of the stud is an advantage as the chances of rapid spread of infection are thereby reduced.

When a mare aborts she should be isolated at once and the ground soiled by the foetus, discharges, etc., disinfected. Mares which have aborted should be kept apart from the rest of the stud for at least six months in an abortion area.

The agglutination test is of value only for the diagnosis of cases due to the B. abortivo-equinus, and as many cases are not caused by this organism its usefulness is limited.

*Avoid congestion.*—If a stud can be split up into small batches of mares located at reasonable distances apart, the chances of rapid spread of the disease, should it be introduced, are considerably reduced.

*Avoid the maintenance of permanent pastures for brood mares.*—Should a case of abortion occur, have the serum tested at once and if positive, all the mares in the stud should be tested.

*Segregate all positive and suspicious reactors in separate batches.*—Retest non-reactors to the first test after an interval of 3 months, again segregating positive and doubtful reactors.

*Retest clean animals at intervals of 6 months to a year.*—Vaccination with a sterilised vaccine has been practised in different countries with varying success. It is recommended by Edwards in this country as offering a reasonable chance of combating the disease but up to date there are no very convincing results which would warrant one being too sanguine as to the success of this method. Under certain circumstances, however, it is well worthy of trial as being the only method which offers any hope of success.

NOTES.

## CONTAGIOUS BOVINE PLEURO-PNEUMONIA

**Nature of Disease.**—A specific contagious disease of cattle, the lesions of which are confined mainly to the lungs and pleura, and characterised by a profuse exudation of sero fibrinous lymph into the interstitial connective tissue of the lungs and into the pleural cavity.

**Susceptibility.**—This particular form of pleuro-pneumonia is practically specific for cattle, but buffaloes, camels and deer are said to be susceptible. Calves under 2 months old are less susceptible than older animals.

**Prevalence.**—Until recently the disease was thought to be non-existent in India, but outbreaks have occured in Assam, and two strains of virus from Assam have been established in hill bulls by the Imperial Veterinary Research Institute, Mukteswar. It is therefore considered that a brief description of the disease is necessary. The disease has at different periods ravaged most of the countries of Europe, also the continents of Africa, America and Australia. It prevailed over a great part of Europe, including Great Britain, throughout the greater part of the 19th Century, but by 1910 had been successfully eradicated from European countries with the exception of Russia and Spain. The United States became free in 1892, and Great Britain in 1898. The disease still exists in Australia, parts of Africa and in Asia.

**Bacteriology and Infection.**—The causal organism is very minute, and only just visible as a refractile speak under the highest powers of the microscope. It is able to pass through a Berkefeld V or Chamberland F filter, which filters are capable of arresting all ordinary visible bacteria. It was first cultivated in 1898 by Nocard and Roux. They inoculated nutrient broth contained in collodion ampoules with the lymph. These ampoules were then introduced into the peritoneal cavity of rabbits. By this means the virus is supplied with nutriment through osmosis which readily takes place, but is protected against the action of leucocytes. After 14 days the broth is found to be slightly hazy and on microscopical examination is found to contain numerous small refractile bodies appearing as mere points. These bodies are too small for any definite form to be determined. Other methods of cultivation have since been devised on special media without the intervention of rabbits. The organism has also been demonstrated to be pleomorphic, occurring in artificial media as refractile points, short spirillæ, asteroid bodies and branching mycelia. The organism has now been placed in the order Borrelomycetales, and has been named Borrelomyces peripneumaniae.

Fatal disease in cattle is set up by the inoculation of fresh lymph from an affected animal or by pure culture. There is an intense local reaction with considerable swelling and the exudation of lymph into the subcutaneous and intra-muscular connective tissue, resulting in a marbled appearance of the affected muscles similar to the appearance seen in the lungs of naturally affected animals.

Animals which recover from such inoculation have acquired an active immunity both against the natural disease, and experimental infection. The lung lesions of the natural disease cannot be produced by experimental infection. Attempts to reproduce the natural disease by drenching, and other methods, have failed. The natural method of infection, therefore, is not determined. Direct contact of diseased with healthy cattle is the most certain method of infection. There is a difference of opinion as to how long the virus can remain active and infective outside the body in cow-sheds, etc., some even asserting that there is no danger in allowing healthy cattle to occupy sheds that have been recently occupied by affected animals; but it is generally agreed that the biggest danger is an affected or recovered animal.

The virus remains active in affected lung tissues for many months, and pure cultures can be kept virulent for about 10 months or more under certain conditions. The natural method of infection is probably by inhalation. An animal that has apparently recovered from an attack may retain encapsulated and necrotic foci in the lungs—the so-called "lungers", and such animals are recognised as the greatest source of danger in introducing the disease into a herd.

**Symptoms and Diagnosis.**—The period of incubation in natural cases is about 14 days. The early symptoms may be indefinite, consisting of dullness, loss of appetite, suspension of rumination, etc. In a milch cow the yield of milk falls. The temperature is raised 2° or 3° and the respirations are accelerated. Later the animal develops a cough, after which the usual symptoms of pleurisy and pneumonia may develop rapidly. Auscultation will reveal hepatised patches, and possibly fluid in one or both sides of the chest. If the disease is acute, the symptoms become aggravated, and the animal usually dies in from 10 to 20 days. In quite a number of acute cases, the acute symptoms subside and the animals appear to recover gradually returning to an apparently normal state of health, and improving in condition. Such animals may retain encapsulated and necrotic foci in their lungs for long periods, which are only discovered on *post-mortem*, and it is these animals that remain, during their life, a potential source of infection. Other acute cases do not make an apparent complete recovery; but the symptoms become chronic, and the animals become wasters suffering from a chronic cough and debility, which may end in death after a varying period. This type of case is a more dangerous source of infection than the apparently recovered "lunger", as the lungs remain more extensively affected and the chronic cough is a means of disseminating infective material.

Other types of cases met with are the hyper-acute, with rapid death; and abundant outpouring of pleuritic exudate; and mild abortive cases, which only show a rise of temperature with rapid recovery.

In cases in which hepatisation of the lung has taken place it is doubtful whether complete resolution ever takes place. From what has been said above it is obvious that the disease might be kept going in a large herd for years through the existence of lungers, and the introduction of fresh stock. The diagnosis of the disease is readily confirmed on *post-mortem* examination.

**Lesions.**—The lesions in the thoracic cavity are very characteristic. There is a variable amount of sero-fibrinous exudate in the pleural cavity, in some cases as much as 2 or 3 gallons. In other cases although the pleura is inflamed there may be practically no exudate present. In almost all cases the visceral pleura is covered with a layer of yellow gelatinous looking fibrinous lymph of variable thickness, and the pleura itself is infiltrated with serous exudate. The pericardium may shew similar changes. The most characteristic lesion is that of the lungs. There is more or less extensive consolidation of one or both lungs and the consolidated areas are swollen and stand out from the surrounding unaffected areas. On cutting in to the affected areas a clear straw coloured lymph oozes out in large quantities. This may be blood-stained and tends to coagulate on standing. The cut surface of the lung has a very characteristic marbled appearance. This is due to the thickened interlobular connective tissue septa. These septa which normally are very thin and hardly visible to the naked eye, become infiltrated with the same straw coloured lymph until they may be enormously distended and their connective tissue thickened. The islands of lung tissue surrounded by these thickened septa vary in colour and may be rose, pink, yellow-orange, bright-red and dark-red to nearly black, this variation in colour being due to the inflammatory process affecting them and the age of the lesion. It is this variation in colour of the lung tissue and the vein-like disposition of the connective tissue that give such a striking and characteristic marbled appearance. The bronchial and mediastinal glands are swollen, soft and oedematous.

In chronic cases the pleura is almost invariably involved and there are adhesions between the parietal and visceral layers, and less free fluid in the chest. The pleura is thickened through the formation of inflammatory fibrous tissue. Also the interlobular septa of the lungs, the thickness of which in the first instance was due to serous infilteration becomes firmer from the formation of inflammatory fibrous tissues, less fluid oozes out of the lung on section.

In most cases of the disease, areas of necrosis are to be found in the affected parts of the lung, due to thrombosis and to the toxic action of the virus. These necrotic areas are paler in colour and gradually become encapsulated, forming sequestra. In a recovered animal these sequestra are a potential source of danger as they may contain living organisms, and if broken down become a source of infection to other animals.

The microscopical characters of the lesions are what would be expected and there is no need to go into detail. The changes in the parenchyma of the lung are evidently secondary to those in the connective tissue and the pneumonia is of the croupous variety.

Diagnosis is not always easy at the outset, since the clinical symptoms are simply those of pleuropneumonia. However, as in a herd, it would be exceptional to get an isolated acute case, the diagnosis is rendered easier. If several cases of pneumonia occurred in a herd in an area in which contagious pleuropneumonia was endemic, one's suspicions would be immediately aroused. A positive diagnosis can only be made by *post-mortem* examination, and it would probably be advisable to destroy one of the worst affected animals for this purpose, to enable preventive measures to be adopted at the earliest possible moment. Given an outbreak of the disease, there are no other diseases with which it could be easily mistaken. An isolated case might be mistaken for such diseases as tuberculosis, traumatic pericarditis, or sporadic pneumonia.

### How to deal with an outbreak.

**Immunisation.**—The oldest method of immunisation and one still largely practised in countries where the disease is endemic is Willem's method. The material used for vaccination is the fresh lymph from the lungs of an animal just dead of the disease or one that has been killed at the height of attack. The lymph should be extracted from portions of the lungs shewing characteristic hepatisation and marbling and not from necrotic area. This is done by incising deeply, when the clear amber coloured lymph rapidly exudes and is collected in a sterile vessel. This lymph should be used immediately and 2 or 3 drops are introduced under the skin near the tip of the tail. This may be done by means of a strong hollow needle or by passing a piece of narrow tape soaked in the lymph under the skin or by means of a syringe. The reaction takes place in from 6 to 20 days, and a few days after its subsidence the animal is immune to experimental infection with either lung lymph or pure culture. Occasionally accidents happen as the result of inoculation. Gangrene of the tail may be set up and the tail may drop off as the result. If the inflammation extends forward a fatal result may ensue.

Lymph that is not required for immediate use may be preserved by the addition of one volume of glycerine and one volume of 5 per cent. phenol to two volumes of fresh lymph. Such lymph remains active for 2 or 3 months. Another method of vaccination is Pasteurs. In this method the lymph is obtained by inoculating a calf 4 to 6 months old with virulent lymph in some region where there is plenty of loose subcutaneous tissue, *e.g.*, behind the point of the elbow. This causes a large swelling as the result of infiltration with lymph. The swelling, when at its maximum, is incised, and the lymph is collected and used in exactly the same way as in Willem's method. It is said not to give such a strong immunity

as lymph from a natural case of the disease. Another and more recent method is Nocard's, in which a pure culture of the organism is used as the vaccinating material. A hyper-immune serum can be produced, but it gives only a brief immunity: as large doses, of the hyperimmune serum are required, it is expensive and of little practical value.

In European countries the disease has been tackled in different ways. It has been proved possible to stamp it out by drastic measures of slaughter, segregation, and vaccination. In less civilised countries, such as the Sudan, one could not hope to do more than keep the disease under control by slaughter of obviously affected in a herd and vaccination of the remainder.

The lines of procedure which may be adopted are:—

(*a*) Compulsory slaughter of all affected animals together with all in-contacts and those suspected of being infected.

(*b*) Compulsory slaughter of the affected only, with immunisation by inoculation of all animals which have been in contact with affected animals, or are in a declared infected area.

It was by the first of these methods that the disease was stamped out in Great Britain. In Australia a modified method is adopted, *viz.*, slaughter of affected, inoculation of in-contacts and of cattle on neighbouring holdings, and quarantine of all in-contacts on the farm, these latter only being removed to an abattoir for slaughter under licence.

NOTES.

# NOTES.

## CONTAGIOUS PNEUMONIA OF THE HORSE.

**Nature.**—This disease was formerly confounded with Equine Influenza. It is an infectious low type of pneumonia usually affecting both lungs with complicating lesions of the pleura, heart, pericardium, liver bowels and kidneys.

**Bacteriology and Infection.**—As in influenza, no specific organism has been definitely proved to be the cause of this disease. The cause is held by some to be a filterable virus in association with which are secondary invaders, *viz.*, Pasteurella and Streptococci (the Streptococcus or Diplococcus, of Shutz). The virus, however, is of a less diffusable and more fragile type than that of influenza. There are several accessory causes, *viz.*, youth, debility, inclemency of the weather, exposure, the presence of nasal or bronchial catarrh, close foul air, bad ventilation, overcrowding, underfeeding, overwork, exhaustion, long journeys by rail.

The infection clings to yards, stables, mangers, troughs buckets, litter and manure; convalescent horses may carry it for weeks.

It spreads much less rapidly than influenza and tends to stick to certain stables. Infection has little disposition to spread beyond the stable into which it is introduced or the near vicinity of the diseased animals. Incubation is from 3 to 10 days.

**Symptoms and Diagnosis.**—In a unit the disease usually follows the introduction of fresh young horses, or remounts. This fact, the appearance of several cases either at the same time or in succession, and the appearances on *post-mortem* examination are our guides in diagnosis. The pneumonia is almost always double, and in isolated areas in the lungs. This is almost diagnostic of the disease. Crepitation may be heard round the border of the consolidated patches. Symptoms of pericarditis and endocarditis are especially common in the more severe types of the disease and may be suspected by the soft weak or imperceptible pulse or tumultuous heart beats.

A yellowish discharge from the nose with a tendency to dry into a yellow crust around the anterior nares is an almost constant sign; the conjunctival, and to a less extent the buccal mucous membranes shew a yellow shade, early in the disease, and the colour may even be brown or orange.

The urine is always scanty, high coloured, and usually albuminous when the disease is at its height. Swelling of the legs and dropical swellings are indications of congestion of the liver which is usual in the disease. *Post-mortem* examination shows the double character of the pneumonia, with circumscribed areas of consolidation especially common near the lower border and anterior part of lungs. Each area of consolidation shews a purulent or necrotic portion with a surrounding zone of dark red congestion. Purulent sacs containing gangrenous masses are common. Black areas of infarction, the beginning of the gangrenous process, are to be seen. Pleurisy is usual over the consolidated areas. As these are commonly

circumscribed, an excessive hydrothorax is exceptional but at times it is abundant. The enlargement of the congested liver is a marked feature, the weight of that organ frequently attaining 30 lbs.

The above *post-mortem* appearances quite distinguish it from ordinary pneumonia; and the disease is distinguished from Influenza by the absence of the great nervous prostration, the eye symptoms and by the less rapid progress of the disease.

Moderate cases may last from 2 to 3 weeks, convalescence being completed by the end of the 3rd or 4th week. Death rate is usually high at the outset and varies from 1 to 20 per cent. The disease may leave in its train such affections as roaring broken wind, cardiac insufficiency, disease of joints or tendons, liver trouble.

## How to deal with an outbreak.

1. **Isolate.**—If a portion of the hospital cannot be specially set apart as an isolation ward, send case or cases well away from the lines, placing under shady trees, or build chapper protection.

2. Isolation must be complete,—separate attendants, watering and feeding arrangements, and nothing to be used in common with healthy animals.

3. Destroy animal if no hope of recovery. Burn carcase, clothing, etc. This reduces the centre of infection.

4. Disinfect thoroughly affected standing and one on each side, burning all broken fodder and bedding, and all bedding that may have been mixed or come in contact with that of affected animals. Disinfection measures should be specially directed to the manger, wall in front of manger, manure, drains, common water-trough, or anything likely to have become contaminated by discharges from the nose. If several cases have occured in a stable, vacate it for at least a fortnight and thoroughly carry out disinfection measures.

5. Keep a strict watch for at least 10 days on animals of the same troop of stable, or that in any way have been exposed to the infection, isolating at once any suspicious case shewing fever. Take the temperature of each animal every morning so as to diagnose cases as soon as possible and before work has aggravated the disease.

6. **Treatment.**—Attend to nursing and hygienic measures, fresh air, warm clothing. Pure water changed often, linseed tea, and barley water are of importance in allaying thirst. Milk is less liable to produce pleuritic effusion. Inhalations of carbolic acid or other antiseptics vapourised from hot water are useful; Salicylate of soda or hyposulphite of soda are good internal antiseptics. Stimulants are not always satisfactory. Treatment should be in accordance with severity or otherwise of the case. In severe cases applications of hot rugs to chest do good and when there is a tendency to pleuritic effusion, iodide of potassium is beneficial, and tapping

the chest should be resorted to. In all cases the patient should be kept in hygenic atmosphere and surroundings. Intravenous injections of 3 grammes neosalvarsan in 60 c.c. of distilled water have given satisfactory results.

7. Convalescent animals should not rejoin their units for at least six weeks.

**Prevention.**—Newly purchased animals specially from dealers' stables, where the disease is liable to have a permanent abode, should not join a unit or stable before segregation of one month.

# NOTES.

## CONTAGIOUS STOMATITIS

**Synonyms.**—Stomatitis Pustulosa Contagiosa.

**Nature of Disease.**—A contagious eruptive inflammation of the mucuous membrane of the mouth and lips characterised by fever and eruptions on the affected parts. For the most part it is a benign disease occasioning very little if any noticeable constitutional disturbance; but under certain circumstances, to be mentioned later, may assume a virulent and acute form, which spreads rapidly and may be the cause of considerable, though temporary, inefficiency in a unit or formation.

**Susceptibility.**—Commonly affects horses for which it is probably specific. Other species of animals are subject to forms of contagious stomatitis, but the casual agent varies, and it is doubtful if it is ever the same as that of the specific disease in horses. Cases have been reported affecting the hands and face of stablemen attending infected animals.

**Prevalence.**—The distribution of the disease is probably world wide. In India it is not of much economic importance. Cases are reported from time to time either in sporadic form or as a mild outbreak. Such an outbreak occurred in Secunderabad amongst the Cavalry horses at the end of 1921 and beginning of 1922. A large number of animals were affected but the disease was of a very benign nature and occasioned a minimum of inefficiency. Like other contagious diseases it may assume importance under certain circumstances, *e.g.*, active service conditions when large numbers of animals are collected together and are living under adverse hygienic surroundings.

**Bacteriology and Infection.**—The causal organism has not been demonstrated and is probably a filterable virus.

Infection is transmitted by means of saliva, and as an animal in the acute stage of the disease dribbles quantities of saliva, infection can be very rapidly spread by means of contaminated fodder, drinking water, utensils, etc.

The period of incubation is 3 to 8 days, and the duration of the disease 10 days to 3 weeks, depending on the severity of the lesions.

**Symptoms.**—In the initial stages there is some constitutional disturbance marked by a rise of temperature and accelerated pulse. This stage may pass unobserved and the first symptoms to be noted may be dribbling of saliva caused by soreness of the mouth, examination of which will disclose areas of inflammation and eruptions.

During the eruptive stage the animal may be off its feed. The eruptions take the form of small hard nodules up to the size of lentils. These may be situated on the inner aspect of the lips, the gums, the inner surface of the cheeks or under the tongue. They may also extend to the skin and lips, the wings of the nostrils and the nasal mucous membrane. When the latter is affected there may be a slight muco-purulent discharge. Lesions are rarely seen in other parts of the body. After 3 or 4 days the nodules become

pustules which burst after a few days and form ulcers. Lesions on the skin form scabs under which healing takes place.

*Differential dignosis.*—This benign form of stomatitis might be mistaken for horse pox but in the latter disease lesions are found behind the pasterns as well as in the mouth. In addition to this benign form of contagious stomatitis there is another form which presents an entirely different clinical picture. It is more of the nature of a vesicular or aphthous stomatitis and the lesions are confined to the mucous membrane of the mouth. It is a much more acute disease and is characterised by acute inflammation of the mucous membrane of the lips, gums, cheeks and tongue with necrosis and shedding of large areas of mucous membrane. This acute form of stomatitis was very common in France during the Great War and thousands of animals became affected. The symptoms are more or less the same as in the benign form, except that they are more marked and the constitutional disturbance is greater. An animal may be off its feed for several days due to extreme soreness of its mouth. There is usually profuse salivation and smacking of the lips. If the mouth is examined, it is found to be very hot, the mucous membrane acutely congested, and if the disease has been in existence several days, red raw areas may be seen on the lips, cheeks or tongue where the mucous membrane has been completely shed, or white patches of necrosed unshed epithelium. The commonest site of these lesions is the tongue, and frequently as much as half the upper surface of the tongue may be denuded of epithelium, or the whole of the tip of the tongue. There may be similar patches on the inside of the lips. In milder cases there may be smaller discreet ulcers on the gums and lips. After shedding of the mucous membrane healing is rapid; but in severe cases complete recovery may take several weeks.

**How to deal with an outbreak.**—Isolate the affected and in-contacts immediately and carry out routine measures of disinfection. A daily inspection of the mouths of all animals of a unit should be made, and any showing signs of inflammation should be isolated. A common water trough is a fruitful source of infection and when the acute form of the disease has made its appearance it may be expected to spread rapidly through a unit. Particular attention should be paid to the disinfection of mangers, pails, stable floors, fodder and anything that may have become contaminated with saliva which is shed so profusely.

In the early stages animals should be kept on sloppy food being disinclined to masticate solids. The mouths of affected animals may be douched with any mild antiseptic lotion. Sulphate of magnesia in the drinking water is a useful febrifuge. No drastic internal treatment is indicated.

Healing is rapid & in mild cases no
cicatrices are left.

NOTES.

**Synonyms.**—Epizootic Paraplegia; Paraplegia.

**Nature.**—An acute specific disease of Equines, recently recognised in India and characterised by nervous derangement varying in intensity from violent paroxysms to a mild in-coordination of the locomotory apparatus.

It is not contagious in the ordinary acceptation of the term, cases showing no apparent relationship to previous ones. Cases of actual incontacts developing the disease are rare.

**Distribution.**—In India several outbreaks of an enzootic nature have been recognised, mainly in the North-Western portion of India, but also in Eastern Command and Bombay District. The disease appears to affect horses only. On the plains outbreaks occur from the commencement of the cold weather and subside with the onset of summer. In the hills outbreaks have occurred towards the end of the monsoon period.

**Etiology and Infection.**—Nothing is definitely known with reference to the etiology of this disease in India, but there are many points of resemblance to the Encephalomyelitis of Equines which occurs in America. In America where the disease is known to be caused by a virus, the virus has been demonstrated in assassin bugs (Triatoma sanguisuga) in Kansas. While the distribution and bionomics of the species indicate that this bug is probably not the chief vector of the disease among Equidae in that country the finding may prove significant as an indicator of possible reservoirs for the virus among the many species which may be attacked by the bug. The American disease has been readily transmitted experimentally by ten species of mosquitoes (genus Aedes) and by the Rocky Mountain spotted fever tick (Dermacentor andersoni). It has been produced experimentally by intranasal inoculation of material from affected horses, the incubation period by this method being about 8 days.

The existence of "carrier" cases is suspected and horses which have recovered from one attack are reported to have developed a second fatal attack about a year later. The disease is observed in summer.

**Symptoms.**—The disease usually attacks animals in good condition, and has so far been reported most frequently, if not entirely, in imported horses or in horses with a larger percentage of imported blood. In some cases a staggering gait and incoordination of movements of the hind quarters may be noticed, but in others more alarming symptoms may appear quite suddenly. In such cases the animal falls to the ground, is unable to rise and struggles in a convulsive manner causing severe external bruising and lacerations especially about the head, and chiefly in the region of the zygomatic ridge. There is partial or complete paralysis of the hind limbs and in severe cases the paralysis may extend to the fore-limbs. Cases showing these severe symptoms suddenly, usually terminate fatally in one—five days.

In the majority of cases, the first of the clinical symptoms to b observed may be (*a*) Lack of co-ordination of the hind quarters (*b* Staggering gait (*c*) Stiffness in gait. These indications are bes observed when horses are first led out in the morning and especiall after a cold night. On turning the animal "about" there is marked sinking of the loins, often accompanied by crossing of th hind legs. The hind toes are dragged at the walk and sometime crossing of the fore legs is also observed. The animal often hit himself in progression. There may be exaggerated extension o abduction movements of one or both fore limbs.

In contradistinction to the American disease, fever is rarel observed as a primary symptom in animals which develop partia or complete paralysis. Fever has, however, been observed i apparently otherwise healthy horses in the same unit or part of unit concurrently with paralytic cases. The relation of these feve cases to the actual disease is obscure. The visible mucous membrane are slightly congested, and may be icteric; the tail is nearly alway lifted; relaxation of the sphincters and, in the mare, excoriation o the skin on the inside of the thighs due to incontinence of urin may be observed. In severe cases there may be complete retentior of urine. Paraphymosis in the male due to paralysis of the re-tractor muscles may occur. The lower lip may be pendulous. Swel-ling of both hind fetlocks is almost a constant feature. The appe-tite is unaffected except in severe cases. Hyperaesthesia, often pro-nounced may sometimes be observed in the early stages of an attack In the last stages spasmodic twitching of the pectoral and neck muscles is generally observed, and an animal often dies in a comatose condition.

**Post Mortem.**—Lesions are neither striking nor numerous, but when taken together with the previous history may be of value in arriving at a diagnosis. Cystitis, which may vary from an acute general hæmorrhagic inflammation to a few petechiae, is always present and the contained urine is usually altered in character. A mild degree of gastro-enteritis in patches is usually present. The most typical lesion of the disease is the congestion of the vessels of the meninges with the presence of punctate hæmorrhages in the brain substance. Hæmorrhages may also be found in the kidney. For laboratory examination pieces of brain and spinal cord and, where affected, pieces of internal organs should be sent in preser-vative for histo-pathological examination. Whenever possible serum and cerebro-spinal fluid should be collected aseptically and forwarded for biochemical or biological examination.

**Differential Diagnosis.**—In the absence of a precise etiology differential diagnosis is difficult, but the disease described here may be differentiated from Azoturia by the history—sudden onset after starting work—profuse sweating, tenesmus of crural muscles and coffee coloured urine. At *post-mortem* the boiled or fish meat appear-ance of the lumbar muscles and degenerative changes in the kidney

are characteristic......It might also be confused with heat stroke but this is more commonly seen in the hot weather and is marked by abnormally high fever, weak rapid pulse and acute respiratory distress. At *post-mortem* the oedema of the lungs and general passive congestion will help to establish diagnosis. Similarly with Anthrax, in which smear examination of the blood or œdematous swelling will reveal the causal organism. In the case of Kumri differentiation is difficult in some cases but the usually sporadic occurrence of this disease...........the absence of change in the urinary system, and the absence of congestion of the spinal meninges or cerebral hæmorrhage on *post-mortem* may serve as a guide.

**Treatment.**—All animals showing pronounced lack of control or unsteady balance should immediately be placed in slings. The following routine treatment is recommended.

(*a*) Saline mixture consisting of Mag. Sulph. ounces fourteen. Pot. Nit. drachms two in a liberal quantity of luke warm water per stomach tube.

(*b*) Pass the catheter. In all but the mildly affected the bladder is invariably found full.

(*c*) Hexamine in six drachm doses dissolved in two ounces of sterile water and given intravenously for four consecutive days was thought at one time to have a beneficial effect, but later experience has not borne out this belief.

Recently, however, Sulphanilamide has been used experimentally in the treatment of this disease and the results have been promising. It should be given for the first 4—5 days from the onset of the disease, and should be combined with symptomatic treatment and forced exercise.

(*d*) Symptomatic treatment as indicated in severe cases.

(*e*) Exercise is strongly recommended. If necessary, forced exercise, with the assistance of four or five men to support the animal, should be resorted to.

(*f*) When violent convulsions, accompanied by paralysis of the fore limbs are present or when the animal is unable to support itself in slings—treatment is of little avail.

**Course.**—Varies with the degree of severity of attack and may take anything from two weeks to three months.

**Prognosis and Mortality.**—As a rule those cases showing slight symptoms when first detected are fairly certain of recovery under treatment.

Mortality is high in cases which collapse suddenly and are unable to support themselves when placed in slings.

In an outbreak mortality rate is about 20 to 25%. Complete recovery, frequently delayed, occurs in 60 to 70%. Those cases which do not completely recover exhibit varying degrees of motor ataxia of the hind quarters, which in some cases interferes with the future serviceability of the animal.

**Control.**—In an outbreak of Equine Encephalomyelitis, the principles of action adopted in dealing with contagious diseases should be observed.

1. Treatment of affected in isolation.
2. Daily inspection of other animals in the unit.
3. It is considered that in a large percentage of cases there is a preliminary phase of pyrexia which returns to normal before the onset of actual symptoms. It is therefore, recommended that the systematic taking of temperatures morning and evening of all animals in the unit should be instituted. Those exhibiting a rise of temperature should be regarded as "suspects", isolated, treated and kept under observation.
4. For practical purposes the unit may be declared free three weeks after the last case although fresh cases may appear after that period.
5. It is worth mention that in the U.S.A. chick embryo vaccine has been used with success to confer immunity from the American strains of the virus.

# NOTES.

**Nature.**—Epizootic lymphangitis is virulent, inoculable disease, characterized by enlargement and suppuration of the superficial and subcutaneous lymphatic vessels accompanied by the formation of pustules which later develop into ulcers.

**Susceptible animals.**—The disease is observed almost exclusively amongst horses, mules and donkeys. It has been reported amongst cattle in Japan.

**Distribution.**—It is fairly common in India, and it is important to note in connection with countries from which animals are imported into India, that it exists in China, Italy, Egypt, South of France and South Africa. There would appear to be none now in the United Kingdom, and none in Germany, America, Australia and New Zealand.

**Bacteriology and Infection.**—The disease is due to a cryptococcus, which is found in the pus and discharge from the ulcers. As many as ten to thirty cryptococci may sometimes be found in pus-cells. It is a slightly ovoid organism, 3 to 4 $\mu$ in diameter, one end generally pointed and the other rounded, and with a characteristic clearly defined contour and refractile double outline. It can be demonstrated, without staining, with a $\frac{1}{6}$th objective, using a No. 3 or No. 4 eye-piece, particular attention being paid to the regulation of the light. In stained specimens, the same power can be used. The following methods of staining are practised:—(*a*) Fix pus by heat, stain with Carbolfuchsin, heating until steam rises; keep adding fresh stain and heating three or four times, wash with water and dry, add Lugol's Solution for two or three minutes, wash with water and dry, decolorize with anilin oil, examine under low power to see when decolorization has gone far enough, remove anilin oil with xylol; examine; if required. methylene blue or Bismarck brown can be used as a counterstain before examining.

(*b*) *Claudius Method of Staining.*—Fix a film of pus in the flame. Stain for 1 minute in 1 per cent. aqueous solution of methyl violet. Wash. Dry. Apply half saturated (0·3 per cent.) aqueous solution of picric acid for 1 to 2 minutes. Wash. Dry. Decolorize with chloroform or clove oil. Dry and examine.

The organism can be cultivated on an infusion of horse dung, agar 1—8 per cent. *plus* peptone 1 per cent. glucose 4 per cent., the tubes being sloped and the surface covered with lymph. The appearance of the growth is dry, wrinkled and scaly. The growth takes place by the cryptococci swelling and throwing off hyphæ. The special medium is necessary for the first three generations, after which growth takes place on ordinary agar, and other media at body temperature.

Infection occurs through wounds, slight or otherwise, the organism from some previous case being conveyed to the wound through the medium of soil, standings, dust, harness, horse clothing, grooming utensils, litter, fodder, parasites, flies, etc., or by the hands, clothes,

instruments, sponges, tow, cotton wool, bandages and other necessaries and appliances used by dressers, etc. An animal may also infect itself accidentally by rubbing or biting.

As a rule the wound heals up and appears to be cured: in course of time, varying with an incubation period of from one to three months, or even six, eight, ten, or more months (average about three months) symptoms of the disease show themselves at the original wound.

Its spread is favoured by collections of animals as in regiments, remount depôts and mule corps, and the numerous wounds usually met with in these units.

**Symptoms and Diagnosis.**—Ordinarily it is a local disease, and shows itself in small nodules or pustules, about the size of a pea, at the seat of an original wound or scar and painful corded lymphatic vessels emanating therefrom. Buds, the size of a hazelnut, form on the course of the corded lymphatic vessels eventually ulcerating and discharging pus at first thick and creamy, and later yellowish, oily and curdy.

The seats of the disease are usually the common seats of wounds and injuries—knees, fetlocks, the inner aspect of legs, head, girth, back and withers. The swollen lymphatics with buds and ulcers follow the course of the vessels towards the lymphatic glands. There is usually no systematic disturbance, and no loss of appetite.

The ulcers shew an attempt at healing, and are characterized by bright red exuberant granulations.

At times the first noticeable sign is diffuse infiltration at the seat of the original wound, without suppuration; or a limb may have the appearance of ordinary lymphangitis.

In some instances (stated to be in 7 to 10 per cent. of cases) there are lesions in the mucous membrane of the nostrils, extending occasionally to the larynx and upper third of the trachea. These begin as small papules, eventually bursting and forming well defined ulcers with raised edges and a dug-out appearance. The same exuberant granulations, met with in ordinary cutaneous cases, are also noticeable in this form. The lesions are usually bi-lateral. If due care is not exercised in diagnosis it may be confused with glanders.

The conjunctiva may be original seat of the disease without any noticeable symptoms externally for some time.

The course of the disease is very slow. Some cases are amenable to treatment, making recoveries in from one to six months; but often the cure is only apparent, and relapses are greatly to be feared, crops of buds and ulcers from imprisoned pus or cryptococci occurring.

**Differential Diagnosis.**—Epizootic lymphangitis, both in its ordinary manifestation and the nasal form, may be confounded with

glanders or farcy. The distinguishing features of the former disease as compared with the later, are, however:—

(1) Healthy appearance generally.

(2) Invariable absence of fever.

(3) Characteristic appearance of the ulcers, which shew a tendency to heal naturally, and comparatively readily on treatment.

(4) Whitish colour and thick creamy consistency of the pus.

(5) Non-reaction to the mallein test, which is diagnostic of glanders, in all its forms.

(6) Invariable presence of the cryptococcus in the pus.

(7) Absence of the diagnostic growth of the glanders bacillus on potato.

(8) Absence of orchitis on inoculation of male guinea pig, which is a phenomenon associated with glanders.

(9) The inconstancy of enlargement of the submaxillary lymphatic glands in the nasal form; the granulating character of the ulcerations, which are not the true characters of glanders; the scantiness of the discharge from the nostrils, and the fact that the ulcerations are more in the lower third of the nasal chambers.

The disease may also be confused with ordinary lymphangitis, spurious forms of strangles particularly about the face, contagious pustular stomatitis with external manifestations on cheek, and bursatti. Ordinarily lymphangitis is an acute affection, attended with high fever, great pain of the affected limb, and there is no suppuration. In spurious strangles, the pus teems with streptococci, but there are no cryptococci of epizootic lymphangitis. The mouth lesions and absence of cryptococci differentiate contagious pustular stomatitis. In bursatti, the ulcer is hard, indurated and full of "kunkurs". In all cases of doubt the presence or otherwise of the characteristic cryptococcus of epizootic lymphangitis should be looked for by miscroscopic examination.

### How to deal with an outbreak.

1. Immediately isolate all affected and suspected animals, isolation to be one mile from their own or other units.

2. Destroy all cases definitely diagnosed and burn, or bury the carcase unskinned.

3. As pus is the dangerous element of the disease, and as the standings, bedding, etc., of the affected and those animals on either side are liable to contamination, causing wound infection, carefully examine animals on either side for wounds, also any animal that has been groomed by the same attendant; if shewing any wound, however slight, isolate as "in-contacts".

4. Carefully inspect all animals of the unit for any further signs of the disease, particularly noting the inner aspects of legs, and places liable to kicks, galls, etc., and not forgetting the nasal mucous membrane and the conjunctiva. As far as possible this should be done daily.

5. Make a list of all animals suffering from wounds (however slight) brushing marks, galls, etc., at the time a case or cases occurred and existed in the lines, and keep a strict watch on them for at least six months. They need not go into isolation for this purpose, but may remain with their unit in working isolation. If, however, they have been actually dressed by the same persons or with the same material or utensils as an infected case in the lines or hospital, segregate in "working isolation," watching them very particularly as "wound in-contacts" for at least six months.

6. Place the whole unit in "working isolation" for six months.

7. Avoid too many isolated groups in a unit as causing inconvenience.

8. By every possible means mitigate the number of kicks. Spread animals out whenever possible, as in picket; place kickers out of harm's way.

9. Institute a thorough enquiry into, and change the system of dressing wounds. Abolish sponges, avoid touching wounds as much as possible either with hand or with syringe, use perchloride of mercury 1 in 500 for some time, restrict the use of cotton wool and tow as much as possible, but keep wounds covered up with gauze or bandages. Use separate pieces of tow or cotton wool for each wound, and burn when soiled. Cover any exposed wounds with dry antiseptic dressing.

10. All wounds, however slight, to be reported. Discontinue practice of dressing wounds in the lines under regimental arrangements: all to be dressed under veterinary supervision. Spray pumps of the simple pattern used for "Flit", etc., are useful by obviating the necessity for hand dressing in a large number of cases where an oily antiseptic dressing can be sprayed on the wound. In Iraq where the disease is very prevalent the use of these as a routine treatment of minor injuries had a marked effect in reducing the number of cases.

11. Keep down flies by every means. To prevent them from molesting wounds, apply cheer pine oil or other suitable fly dressing lightly twice daily.

12. Disinfection must be thorough, and directed against anything contaminated with pus from the abscesses and ulcers. Use heat and fire freely. Certainly destroy all bedding, rubbers, sponges, and clothing of affected. Well burn the surface of the standing or standings, walls, etc., and throw into disuse for at least three months. A brazier's lamp is most useful for disinfection in this disease. Follow the instructions laid down under "Routine of disinfection" but use disinfecting solutions stronger. Carbolic Acid is of little use. Boiling water, chlorinated lime of double strength, and perchloride of mercury as strong as 1 in 250 should be relied on. Disinfection measures should be repeated. Do not forget the clothing of attendants and their boots, which should be burnt if of little

consequence otherwise their clothing should be boiled. Harness and saddlery must have special attention, boiling water, soap and perchloride of mercury being used. In a severe outbreak, disinfect the clothing, grooming kit, stable utensils and saddlery of all animals in the unit even the animals themselves should be thoroughly washed and cleansed. The disinfection of all clothing is a great undertaking, and if no proper means exist in a cantonment for this, apply for special sanction of a zinc or iron bath for boiling purposes.

13. Be most careful to boil any instruments that have been used for opening abscesses, etc., in fact make a practice, in every outbreak, to thoroughly disinfect all veterinary hospital appliances, the hands, clothing, etc., of veterinary assistants and hospital attendants, at once, and to repeat the process during the outbreak.

14. A unit cannot be considered free until six months after the occurrence of the last case. "Wound in-contacts" that have been in segregation and working isolation can rejoin their units after six months.

15. Stencil the letters "E. C." (epizootic in-contact) on the off thigh of all animals with wounds at the time of inspection or present at the date the clinical case was discovered.

# NOTES.

## FOOT AND MOUTH DISEASE

**Synonyms.**—Eczema epizootica, aphthous fever, murrain.

**Nature.**—It is an acute, highly infective, rapidly spreading contagious disease of the lower animals, especially of ruminants, characterized by vesicular eruptions in the mouth, and on the feet and udder.

**Bacteriology and Infection.**—Cattle, buffaloes and goats are chiefly affected, outbreaks in military service being usually seen in the two first species of animals. Pigs, horses, human beings, and fowls may contract the disease from drinking infected milk, eating infected products or by inoculation. It is constantly prevalent, more or less, in all parts of India. The disease can be transmitted to guinea pigs by inoculation of their hind foot pads. Since this discovery, these animals have been extensively used in the laboratory for research work as to the nature of the Virus and immunisation. The danger of using cattle for research work in a country where the disease is non-existent is obvious.

The virus is ultramicroscopic and cannot be cultivated outside the animals body apart from living tissues. The infective material is especially resident in the vesicles. From the mouth, this is distributed by means of the abundant drivelling saliva, on pastures, roads, locations, halters, feeding and drinking troughs, and is readily communicated to healthy stock following in the same places. From the feet, and especially the interdigital space, it is left on vegetation, the ground of locations and other possible media to affect other animals in turn. From the teats it mingles with the milk to infect sucklings, and animals and human beings to whom the milk is given.

Of all contagious diseases there is none where the virus is so easily carried. It is generally distributed by affected animals themselves, but it may be introduced and spread by dogs, vermin, poultry birds and even human beings carrying it on their feet. Infection may also be brought to a unit by bhoosa from infected districts, or by animals bringing bhoosa from these districts.

The latter, and the free movement of all cattle, including stray cattle, along cantonment roads, are usually responsible for outbreaks amongst animals in Military Service. Newly purchased animals from district fairs are frequent introducers of the disease into cantonments. Outbreaks are perhaps more frequent in the spring, during the time of movements to fairs, and the making of bhoosa after the wheat and barley harvest.

Outbreaks on active service are often introduced by slaughter cattle.

One attack does not confer an immunity for more than a few months, so that animals may suffer several times from the malady.

The virus on the whole is of fragile character. The contents of vesicles are rendered inactive by drying at summer temperatures (maximum 88°F.) for 24 hours. A temperature of 158°F. destroys

it in half an hour. Cold tends to preserve its vitality and virulence. Lymph stored in a capillary tube and kept in an ice chest is infective after 14 days; after three weeks it is somewhat inactive, and even after 8 or 9 weeks it proves active on inoculation when large doses are employed. The above are conclusions drawn from laboratory experiments.

Under ordinary or natural circumstances, however, the virus is known to have a vitality of about 18 days outside the body, and French experts consider that in sheds unexposed to the action of sunlight, or undisinfected, it may retain its virulence for several months.

As the result of the disastrous epidemic of Foot and Mouth Disease which occurred in Scotland in 1926 and which was traced to a bacon factory receiving consignments of fresh carcases of pigs from Europe, the Foot and Mouth Disease Research Committee was asked to determine how long the virus of the disease remained active in the different parts of other carcases when submitted to the trade conditions under which they were imported. As a result of this enquiry it was proved that bone marrow from chilled carcases remained active for 42 days, and from the frozen carcases, for 76 days, and that blood from either chilled or frozen carcases remained active for 30—40 days.

In India with its conditions of heat, and more or less open standings, for animals, it is reasonable to suppose that the life of the causal organism outside the body, *i.e.*, on the ground, is perhaps a little less than 18 days, though it may be retained in bhoosa for a somewhat longer period.

Its resistance to ordinary antiseptics is very feeble.

Until our knowledge of the virus is, however, more complete our dealings with the disease must remain more or less empirical, and must continue to follow the lines which the experience of it through ages has taught us. It is certain that the slightest amount of virus is enough to communicate the disease in bovines, which are extremely susceptible.

Infection takes place by—

(*a*) Ingestion—contaminated food and water readily producing the disease. This is probably the chief channel of infection.

(*b*) Inoculation of the mucous membrane of the mouth and the feet by contaminated articles; or the udders of cows by the hands of the milker.

In all channels of infection penetration is effected with extreme facility.

## Symptoms and diagnosis.

The period of incubation of the disease in India is from 24 hours to three or four days, generally thirty-six hours. It is shorter in hot weather than in cold.

The first noticeable symptoms are a shivering fit followed by fever (temp. 103° or slightly over) hot mouth, horns and extremities; a smacking of the lips and salivation. There may be tenderness of the feet as shewn by extension backwards and shaking of the hind feet in turn. Sometimes the first noticeable sign is lameness. About the second day, vesicles 1/3 to 1 inch in diameter appear on the inside of the mouth. The tongue is chiefly affected, but the inside of cheeks, gums and roof of the mouth are also involved. The vesicles burst soon after their formation, exposing a reddish inflammed base with shreds of torn epithelium, which is usually renewed in about four or five days.

At the same time, in cows, smaller vesicles appear on the teats, and if not broken by the hands of the milker, burst in 36 to 48 hours forming sores comparable to those in the mouth.

Still smaller vesicles appear on the feet at the junction of the hoof and the skin, and especially in the interdigital space.

As the malady progresses salivation is more profuse, and lameness is evident.

Sometimes the mouth only is affected; at other times the feet only.

The disease is as a rule a benign affection, and if animals are properly cared for, all symptoms of fever disappear in three or four days, the recovery takes place in from ten to fifteen days with little loss of condition. With common care animals rarely die, but if neglected or worked while diseased, the foot symptoms are aggravated, the hoofs may drop off, the legs become much swollen, abscesses may form in them and death result in 10 or 12 days.

In the pure bred imported cattle, mainly friesians belonging to military dairy farms the symptoms are much more severe than in cattle indigenous to India and in crossbreds. There is a strong tendency for pneumonia to develop as a complication, and the mortality may be considerable, even when the animals are under the best conditions and given suitable treatment.

Many bovines are carriers of Protozoan parasites (Piroplasma, Theileria, Anaplasma and Coccidia), and these may be resuscitated by the concurrent infection and consequent lowering of body resistance. Therefore as soon as a secondary rise of temperature or onset of complicating symptoms are noticed, routine blood examinations should be carried out on the spot and additional blood films sent to the Military Veterinary Laboratory. If the alimentary tract is involved in the complications, a fæcal examination for Oöcysts is essential and specimens of fæces should also be sent to the Laboratory in accordance with the instructions given in Appendix B.

In sheep, the symptoms are similar to those above described but the feet generally suffer most and the animals lose condition. In pigs, too, the disease is almost exclusively localised in the feet, and they suffer much pain.

In human beings there is a tendency to localisation on the same points as in animals. The hands are inoculated in the act of milking,

and the mouth is affected from drinking infected milk and even eating butter made from infected milk. The danger is greatest in children fed on an exclusive milk diet, and who drink it warm. Gastric disturbances of a severe nature in addition to buccal eruptions may result. The latter is mentioned in view of outbreaks in military dairy farms.

**Differential diagnosis.**—The disease is sometimes mistaken for rinderpest, but in foot and mouth disease as found in India purging is not one of its symptoms, whereas diarrhoea and dysentery are invariable accompaniments of rinderpest. In the latter also the feet do not become affected.

### How to deal with an outbreak.

1. Bear in mind the extreme infectivity, and **remove** affected **animals into isolation** as soon as possible. The isolation should be as far removed as circumstances admit, and away from public roads, which on no account should be traversed to reach it.

2. Stop all communication between the isolation picket and the original standings or lines. Isolation must be absolute. Separate attendants are necessary and they should not be allowed to leave the location until the conclusion of the outbreak. Original attendants and milkers in the case of dairy cows should accompany affected animals into isolation. A brushwood or other fence to keep out stray animals is useful. Food for attendants and affected animals should be brought to the location boundary by hand, or if a cart is used, it should be drawn by mules or horses. Visitors should be excluded, and any necessary visitors such as Veterinary Officers, Officers or Non-Commissioned Officers in charge should disinfect hands and boots before leaving, and leave their walking sticks outside, as infection is easily carried on the end of a stick. Loose grain should not be allowed to lie on the ground to attract birds, etc.

3. **Isolate the immediate contacts,** *i.e.*, animals on either side of the affected cases. Wash them with a 2 per cent. solution of Carbolic Acid or other suitable disinfectant, particularly disinfecting the heads, legs, feet and other parts likely to have been contaminated by the saliva of the affected case. Dry afterwards. Strong solutions of disinfectants should not be used, otherwise tender parts may be blistered. In milk cows, the udders should be disinfected: a 4 per cent. solution of Boric Acid is suitable for this. An attendant other than one who has been in attendance on affected animals should be told off to them.

4. **Carefully inspect all animals** for first symptoms of the disease, making free use of the thermometer. Separate any doubtful case.

5. After the above inspection, change the entire standing of the troop or unit, to admit of a thorough disinfection of affected standings or lines. Incontacts will be walked through a foot bath containing 2 per cent. cresol before going to fresh standings and will

be walked through the foot bath four times daily thereafter until the outbreak is terminated. A foot bath for disinfecting purposes can be made with an old tarpaulin placed in a hollowed out portion of ground, if a brick or concrete footbath is not available."

6. **Repeat inspections twice daily,** morning and evening, removing any showing symptoms of the disease to the isolation lazarette for treatment.

7. **Chlorinate water supply** by adding 90 grains of fresh chlorinated lime to every hundred gallons of water.

8. **Enquire into original source of infection.**—It may be possible to prevent its continuance. If bhoosa is blamed, change the source of supply, or give grass: inspect contractor's animals bringing bhoosa to the lines. If contractors take manure away, inspect their animals.

9. **Thoroughly disinfect affected standings** as laid down in "Routine of disinfection" under "General Measures," paying particular attention to the ground, mangers, walls, feeding and watering arrangements (including common water trough), fæces, any halters, line gear, dusters, ropes for tying legs in dairy cattle, poles of carts used by the affected.

Tainted fodder should be destroyed: doubtful fodder may be eaten by mules after being sun-dried for a few days.

Include all other standings in a general clean-up and disinfection.

10. **Treatment.**—Good nursing and hygienic care are essential. The ground on which the animals stand must be kept dry, scrupulously clean, and sanitary; otherwise complications will arise in their feet. In dry weather an open picket is best, and even in rainy weather the shelter of trees will afford dry standings. Failing that, shelter of some kind should be afforded.

Keep the ground on which affected animals stand well treated with disinfectants, and free from flies. Animals with severe feet lesions do well under cover on earthen standings with a good supply of bedding to encourage them to lie down.

Affected cases should be given foot baths containing 2 per cent. copper sulphate. Apply any of the following dressings:—

Eupad (Equal parts of chlorinated lime and boric acid made into a paste with water).

Weak zinc and lead lotion.

2 per cent. Cresol and tar mixture.

Carbolic or boric vaseline (1 in 12).

Similar dressings should be used for the udder and teats, boric applications being the best. Elastoplast bandaging on teat lesions has given good results by preventing secondary infection. Care should be exercised in drawing the milk.

Mouth washes twice daily with a weak antiseptic solution of alum and potasii permanganas followed by dressing of the lesions with a saturated solution of Magnesii sulphas can be given for

buccal lesions. It has been noted that some hours after the application of a saturated solution of magnesii sulphas the salivation in the majority of cases ceases and the animals commence to feed.

During the fever stage give Chlorate or Nitrate of Potash in the drinking water or gruel once or twice daily in half ounce doses.

In order to detect complications, such as pneumonia and septicaemia, etc., routine examination of blood films for the presence of bacterial or protozoan organisms is indicated. This should be carried out as soon as a secondary rise of temperature or onset of complicating symptoms is noticed.

The diet must be of soft green grass such as doob or young lucerne. An abundance of thin rice gruel, in which once or twice a day two or three ounces of goor or treacle are mixed, should be given: this may be alternated with bran mashes in which a little common salt is mixed.

Complications in the feet, such as abcesses, sloughing of the hoof, etc., will require special surgical and antiseptic treatment, covering with cottonwool, tow, bandages and boot. Sulphanilamide is useful in the treatment of arthritis and septicaemic complications and S. U. P. 36 has given good results in treating cases with high temperatures and pneumonic complications (dose: 5 c.c. subcutaneously, 3 c.c. for calves).

Carcases should be disposed of by burial, the skin being first slashed. They should be removed with care, the mouth and feet being well disinfected with strong solution, and the head tied up to prevent drip of saliva from the mouth, or preferably enclosed in sack with a little straw in it. A cart drawn by mules should be used.

The clothing of animals, buckets, line gear and all articles used by the sick animals should receive special attention in accordance with the procedure mentioned in "Routine of disinfection."

The ground in which animals stood must be given a final and thorough disinfection.

11. **Working isolation.**—If the foregoing measures of early separation and isolation of the affected and incontacts, the close inspection of half troop or herd twice daily, the removal of animals from the proximity of contaminated standings and the thorough disinfection of the latter have been carried out, and no more cases have occurred for seven days, healthy animals may perform work in working isolation after that time, up to the date on which the unit is declared free.

Incontacts can return to their lines after seven days clear.

Animals when sent to work in working isolation should first have their feet, especially the interdigital space, dressed with tar.

In spite of all precautions when the disease breaks out in a unit it may get the upper hand and spread rapidly until a large percentage of the animals are affected. In such cases, especially if

the unit is a small one such as a bullock half-troop, it may be a better policy to segregate the whole unit and allow the disease to run through it. By this means the outbreak may be brought to an end sooner and all animals that have become affected will have acquired a certain degree of immunity.

12. **Use of milk during an outbreak in dairy farms.**—The raw milk from animals affected with the disease is not fit for human consumption. The virus however is killed by pasteurisation for 30 minutes at 145°F. Milk therefore can be safely used after pasteurisation provided that—

(*a*) It is not changed in appearance.

(*b*) The animal has no abnormal temperature.

(*c*) There are no lesions on the teats.

13. **When to declare an outbeak at an end.**—A fortnight after the cure of the last case, all recovered animals can be returned to their lines, and the unit declared free from the disease. The precautions with regard to their disinfection and the disinfection of their attendants before return must not be forgotten.

## Prevention of introduction.

(1) Isolation of newly purchased cattle for three weeks, and disinfection of their feet.

(2) Avoidance by purchasing officers of districts or fairs known to be infected.

(3) Avoidance of serais and vicinity of villages when travelling along a road.

(4) Use of Stockholm tar as a foot dressing when districts, known to be infected, have unavoidably to be passed through.

(5) When the disease is found to exist amongst animals other than those in military service in a cantonment, report should be made to Officer Commanding Station and the Cantonment Magistrate, and measures necessary for control, *viz.*, isolation, inspection, restriction of movement, put in force. Sheep and goats in droves should be included in measures of control.

(6) Immunisation by serum and serum-virus has been practised in Europe, but up to date neither has been shown to be of any practical applicability or utility.

A more promising method may prove to be the use of an adsorbed virus vaccine.

# NOTES.

**Nature of disease.**—Glanders-Farcy is a highly contagious disease of the lymphatic system manifesting itself in nodules, ulceration, and degenerations in the respiratory-passages or in the skin, and due to the Bacillus Mallei. When the disease is localised in the respiratory passages it is the custom to term it Glanders: when the lymphatics of the skin are affected, the name Farcy is given to it; but they are manifestations of one and the same disease.

**Susceptibility.**—Practically speaking it is peculiar to horses, mules and donkeys. It is transmissible to man by inoculation. Cattle, chickens, mice and rats are immune; pigs under ordinary circumstances are also immune. Goats, sheep, cats and dogs have contracted the disease, but occurrence in them is uncommon. Guinea-pigs and rabbits are susceptible by inoculation: the former is made use of for diagnostic purposes.

**Prevalence.**—It is not nearly so prevalent in India as formerly, thanks to the most valuable diagnostic agent Mallein, whereby cases can be diagnosed before clinical signs appear. Severe outbreaks are, therefore, seldom or never seen. It chiefly occurs in large cities, *i.e.*, Calcutta and Bombay. It was practically unknown in India before the war of 1857.

It is a disease usually attending war, the debilitating circumstances of active service predisposing to it, and the collection and movement of large numbers of animals making infection more possible.

Australia, Tasmania and New Zealand are up to the present free from the disease, which fact should be noted in connection with the supply of horses from these countries.

**Bacteriology and Infection.**—The Bacillus Mallei is a short slender rod, from one-third to two-thirds the diameter of a red blood corpuscle (2 to 5 $\mu$ long; ·25 to 1·4 broad), somewhat resembling the Bacillus of tuberculosis, but thicker. It is non-motile, aerobic and does not form spores. It is easily destroyed by physical and chemical agents. It is killed in 10 minutes by a temperature of 131°F., in 2 minutes by 212°F. (boiling point), or by corrosive sublimate solution 1 in 5,000, a 5 per cent. solution of carbolic acid or 1 per cent. solution of Permanganate of Potash.

Discharge from the nose, in thick layers, will remain virulent in dry air and ordinary sunshine for two months; in moderate layers for 4 to 15 days; in thin layers 3 days. With the heat of an Indian sun, these times can be much reduced. In water the bacillus remains virulent for 15 to 20 days, and it resists putrefaction from 14 to 24 days. In closed stables, such as in England, it may remain virulent for three or four months. It will not grow in infusions of hay or straw or on horse manure, and it may almost certainly be concluded that it has no saprophytic existence. The control and suppression of the disease is, therefore, very simple.

**Infection is by:—**

(*a*) Inoculation.
(*b*) Inhalation.
(*c*) Ingestion.

The first is the channel of infection in human beings but is infrequent in animals. Care should therefore be exercised in the handling of diseased animals, at *Post-mortems*, and in laboratory experiments, lest infection is contracted through cuts, scratches, abrasions, etc.

In horses, mules and donkeys, infection results from the inhalation of desiccated particles of nasal discharge floating in the atmosphere of stables; but by far the most frequent source is ingestion of the virus in food or water, which has become contaminated with nasal discharge and commonly through the medium of mangers, nosebags, buckets, and water troughs.

Sponges and rubbers are frequent media of infection and spread, the infection being directly conveyed from nose to nose or to the water bucket in which sponges and rubbers are rinsed.

The period between infection and clinical signs of the disease is most variable. Inoculation produces the disease in 3 to 5 days; feeding on virus contaminated food in 1 to 3 months. Many instances are, however, on record where the disease has lain dormant in the lungs for many months. Debility or sickness shortens the incubation period: the same is true of purgation, a strong dose of aloes being the usual practice in days gone by to "bring out the disease" in doubtful cases.

Infection is usually slow, and the majority of horses (more so than mules) seem to have a certain individual degree of immunity or resistance.

## Symptoms and Diagnosis.

The cardinal symptoms of the disease are:—

(a) **Glanders Forms.**—Discharge from one or both nostrils of a glairy sticky nature, snuffing breathing, ulcerations on the nasal mucous membranes, hard tumefaction of the sub-maxiliary lymphatic gland corresponding to the side on which nasal symptoms are observed, and a variable amount of fever.

In acute cases, particularly in mules, the whole face may become considerably swollen, and the respirations of a characteristic wheezing, snuffling or snoring type .

(b) **Farcy Form.**—Tumefaction of the lymphatics of legs, head, neck, or other parts, individual lymphatic vessels standing out in "lines" or "cords" with here and there nodules or "buds" and ulcers along their course ulcers do not show any tendency to heal: they increase in size along the course of the lymphatics and discharge an ichorous yellow pus.

The symptoms vary in accordance with whether the case is acute, subacute or chronic. Both Glanders and Farcy may exist in the case at the same time. In acute Glanders both nostrils are usually affected; the ulcerations, which begin as small greyish nodules with a red or deeper coloured areola, rapidly coalesce, forming ulcerated patches; the discharge is blood stained, and the alae of the nostrils become so swollen that it is difficult to examine the inside of the nostrils properly; the mucous membrane is much congested, sometimes even to a blackish or violet tint, particularly on the septum nasi. The whole septum may become one continuous ulcer leading to perforation. Snuffling is more pronounced; temperature is higher, and Farcy may be present. In chronic or subacute Glanders the symptoms bear a like resemblance to the above, but are less pronounced. The discharge may be confined to one nostril.

Mules and donkeys are more subject to the acute form than horses.

In severe outbreaks it is not uncommon to find symptoms ushered in by acute articular pain in one joint (usually stifle), or dull colicky pain for several days.

Glandered animals of long standing generally look unthrifty and more or less emaciated.

**Differential Diagnosis.**—Though the symptoms of nasal glanders when observed are quite unmistakable, yet it is necessary to differentiate it from other diseases of the upper air passages. For instance in Catarrh and Catarrhal Fever there is discharge from the nose, and sometimes an exfoliation of the mucous membrane at the ala and front part of nostril, but there is no hard ulceration of a chancrous character, and no hard tumefaction of the submaxillary gland as met with in Glanders.

In Strangles there is discharge from the nostrils, and a swelling under the jaw, but the discharge, though yellowish, is not sticky, and the abscess in the submaxillary space enlarges and bursts, discharging pus, which never occurs in Glanders.

In nasal gleet there is no fever, and the discharge can usually be traced to a diseased tooth or maxillary sinus. There is also the characteristic smell of diseased bone, and bulging of the affected part in old standing cases.

Farcy is apt to be mistaken for Epizootic Lymphangitis.

In all cases of doubt the Mallein Test is the great differentiator, giving a reaction in the case of Glanders and Farcy, and no reaction in other diseases.

Inoculation of a male guinea pig either subcutaneously in the flank, or better intraperitoneally, gives a characteristic violent orchitis in two or three days.

The growth of the organism on potato is most characteristic and can be easily carried out. In two or three days drops like honey

appear, becoming, later on, deeper to a chocolate colour. The potato remains unstained.

The microscopical examination of the discharge from nose or from ulcers or pus is not satisfactory. Comparatively few bacilli are present. They stain with difficulty too. Smears are best stained with Methylene blue and then treated with 4 to 5 per cent. Acetic Acid for a few seconds which decolorizes the cells and detritus and leaves the bacilli stained. A magnifying power of at least 800 diameters is necessary.

*Post-mortem* examinations should invariably be made, particularly of the nasal chambers and lungs. In the former will be found different lesions representing different stages, first miliary deposits of leucocytes like grains of sand, then larger pealike nodules made up of nests of leucocytes, and later, ulceration of these nodules, the ulcers being uneven in outline and shewing a tendency to extend in depth and width. The lesions are found on the septum nasi and turbinated bones, and in some cases there is perforation of the septum.

In chronic cases the lungs are usually marked by circumscribed lobular pneumonia and by miliary and larger nodules of degeneration resembling tubercles.

The nodules, which can be felt like shot in the lung tissue, commence as minute points of congestion or ecchymoses, which later, in the centre, shew a translucent or grey mass of lymphoid cells. Later still, this central mass becomes yellowish and caseated, involving the whole area of the nodule. Sometimes no lesions are found in the lungs in animals destroyed under the Mallein Test, but if careful search is made, lymphatic glands will be found affected.

Nodules are also found occasionally in the spleen.

## How to deal with an outbreak.

1. **Destroy all affected animals.**—Though it is stated that recoveries have taken place in the high altitudes of the Rocky Mountains in the U.S.A., where it exists in mild form the disease is to all intents and purposes an incurable one, especially in India, where heat predisposes to quick development. Affected animals must therefore be destroyed, without exception, whether shewing clinical signs or in latent form as evidenced only by the Mallein Test.

2. **Disposal of carcase.**—Can be either burned or buried.

3. **Isolate incontacts on either side of diseased animal.**

4. **Evacuate lines of affected troop or section** pending disinfection and Mallein Test.

5. **Place water trough used by affected animal out of bounds** and note what other animals (troop, squadron or section) have watered there.

6. **Carefully inspect all animals of the unit,** noting in every case the nostrils for ulceration and discharge, the submaxillary glands for swelling, and the body, particularly inside of legs, for swellings, corded lymphatics and ulcers. Inspect healthy troops first, afterwards those in which cases have occurred, and the immediate incontacts last. Use reflected sun from a mirror for inspection of nostrils. With a large mirror worked by an assistant, inspections of a large number of animals are more quickly done. Disinfect fingers and hands with a 5 per cent. solution of Carbolic Acid after every few horses, and certainly after a suspicious case.

7. **Apply Mallein Test.**—This should be done (*a*) as soon as possible to all the animals of the unit, and (*b*) repeated not less than 14 days after to the affected troop or squadron or to animals which in any way have been exposed to the contagion, *e.g.*, the common water trough. The first test informs us if there are any more cases from the original cause, the second disposes of any case resulting from the outbreak dealt with.

Mallein consists of the filtered products of the growth of the Glanders bacillus, and when injected subcutaneously has the property of causing in glandered animals a local reaction or swelling at the seat of inoculation and a rise of temperature. No reaction results in unaffected animals.

**Procedure of Mallein Test suitable for India.**—Use a sterilizable hypodermic syringe, and sterilize by boiling immediately before and after use. Perform the test in the evening between 6 and 8 o'clock after the heat of the day is over. Take temperatures of the animals before the operation and record them. Clip off a small patch of hair, size about 5 inches square, from the near side of neck midway between head and shoulder, clean with brush, and disinfect lightly with a weak solution of Carbolic Acid or Phenyl, without wetting the skin too much. Inject a dose (18 minims) into the centre of the patch, taking care to have the needle well under the skin and not into the skin or the muscles of the neck. At 6 to 8 o'clock next morning again take temperatures, record them, and carefully note if there is any swelling at the point of inoculation. Note also if there is any malaise or lassitude. Do the same from 6 to 8 o'clock in the evening. Visit again the following morning and evening; observe the swelling and take temperatures in such cases. The test may be considered at an end after 48 hours.

In glandered animals a reaction to Mallein consists of a local swelling appearing at the site of inoculation within 24 hours, increasing in size to 36 hours, and persisting until the third or fourth day after inoculation. The swelling is fairly firm, with raised edges, painful to the touch, and in undoubted cases measuring 5 to 10 inches in diameter. Added to this there is a rise of temperature to 104° F., but temperature is not a very reliable guide in India. A large, painful, slowly disappearing swelling with or without a

rise of temperature to 104° F., is an undoubted reaction. In non-glandered animals there is either no local swelling, or it is small, flabby, diminishes after the first 24 hours, and as a rule is gone by the thirty-sixth hour.

Animals should if possible be tested when under cover. When tested in the open a temperature reaction which may be regarded as suspicious may occur in glanders-free animals. It is seldom however that the temperature rises to higher than 103° F., the peak is usually at the 18th hour, and by the 24th hour it has subsided. There is no painful local reaction.

Destroy any cases of positive reaction: keep in isolation any doubtful reactions, *i.e.*, those shewing a small unsatisfactory swelling, or a rise in temperature under 104° F., without swelling, for another test. Retest in not less than 14 days time. If a reaction results, destroy: if there is no reaction return the animal to lines: if again there is a doubtful reaction put back for another test or refer to the A. V. O., Command for further instruction. If on third test the reaction is still doubtful, destroy: if no reaction, return to lines. Many cases of doubtful reaction will be encountered, and one has to exercise one's judgment and experience in such cases. Do not give the benefit of the doubt in repeatedly doubtful reactions, but destroy as suspicious. In doubtful reactions use a double dose, especially in mules.

Another reliable method of applying the Mallein Test, and one which was used extensively during the Great War, is the intradermo-palpebral method. In this test 2 or 3 drops of special concentrated mallein are injected intra-dermally into the lower eyelid about a quarter of an inch below its margin towards the inner canthus. A positive reaction consists of swelling of the eyelid, which commences about 4 to 6 hours after injection, and increases in size and intensity up to 48 hours and usually persisting for several days. In addition there is usually some infiltration of the surrounding subcutaneous tissue, with partial closing of the eye, and a copious mucopurulent discharge from the inner canthus, with acute conjunctivitis.

In a non-glandered animal there is usually no reaction whatsoever, but there may be a certain amount of swelling of the lower lid within a few hours of the injection, but this swelling entirely disappears within 24 hours, and it is not accompanied by any conjunctivitis or discharge from the eye.

Any reactors or doubtful reactors should be retested subcutaneously without delay.

This test has a very great advantage over the subcutaneous test when large numbers of animals have to be tested, as it can be carried out very rapidly, and in the vast majority of cases, the result can be told in 24 hours. Also either eye can be used.

A third method of testing with mallein is the Ophthalmic Method in which concentrated mallein is instilled into the conjunctival sac.

The reaction is similar to that in the intra-dermo-palpebral method, but is stated to be not so reliable.

The Agglutination and Complement Fixation Tests may also be employed for the diagnosis of Glanders.

8. **Disinfection.**—Carry out "Routine of Disinfection" as given under heading of "General Measures," paying particular attention to mangers, nose bags, rubbers, head collars and ropes, flooring, bedding, buckets, water troughs and anything likely to have become contaminated with discharge from nostrils or ulcerations on body. As the bacillus does not form spores and is easily killed, one disinfection thoroughly applied will suffice.

Burn any broken fodder, food in mangers, and bedding of animals affected and of two animals on each side; deal with that number of standings, entirely demolishing the mangers, and thoroughly scraping, washing with boiling water and afterwards disinfectant, the wall and pillars in front of the mangers. Destroy nose-bags, head collars and head rope, clothing, sponges, and rubbers on any shewing of clinical nasal symptoms. The clothing and line gear of the incontacts on either side should be throughly disinfected, or destroyed in case of doubt. As nose-bags are liable to get mixed, or to have been in contact with each other during issue of rations, thoroughly disinfect the whole lot belonging to affected troops, squadron, or section.

After disinfection, standings should be exposed for a fortnight and renewed after that time.

Do not forget the syce and his jharons in the process for disinfection.

Carefully disinfect water trough if used by animal with clinical nasal symptoms. After disinfection expose to action of sunlight for at least fourteen days. Do not fill with water during that time but keep perfectly dry.

9. **When to declare outbreak at an end.**—Without mallein, a unit could never, with confidence, be declared free from glanders owing to the possibility of the existence of latent cases which might develop into clinical cases many months after the outbreak was apparently over. With mallein the unit is free after all animals have successfully passed the test, *viz.*, after the second test of the affected troop or incontacts not less than 14 days after first test.

Affected troop or sub-section and the incontacts can return to the lines after passing the second test, and after disinfection process is complete.

10. **Work and working isolation.**—Work need only be stopped during the two days of the Mallein Test. The affected troop squadron, sub-section. or section as the case may be, should undergo working isolation until after second test

**Prevention of Introduction.**—All horses and mules purchased in countries or districts in which the diseases is known to be prevalent

should undergo the Mallein Test before being admitted to unit lines. This test should be particularly applied to horses and mules coming from a country where war has recently existed.

**Glanders on Active Service.**—Glanders usually following in the wake of war, Veterinary officers and Veterinary Assistants should be ever on their guard against it. A system of periodical inspection should be adopted in every unit. Cases with their line gear should be immediately isolated and destroyed, and arrangement made particularly for the cleaning and disinfection of the feeding and watering utensils of affected troop, squadron or unit, some of which have to be shared by several animals. The Mallein Test should be applied in the field when opportunity occurs, and no grace should be given to reactors or doubtful cases. Careful vigilance, quick action, simple but complete measures go a long way to keep the disease in subjection.

The test should also be applied to all animals brought from overseas whether the country from which they come is reputed to be free from the disease or otherwise, as infection may occur through the medium of ships having carried glandered horses.

**Synonyms.**—Pasteurellosis; Malignant sore throat; Ghotu, Golghotu and Ghotwa (India); Barbone (Italy).

**Nature.**—It is an acute, infective blood disease, due to a cocco-bacillus, and characterized by sudden onset, rapid and very fatal course, marked hyperthermia, extensive gelatinoid or sanguineous extravasation in the intermaxillary space, tongue, skin, subcutaneous and intermuscular connective tissue, lungs, pleura, heart or stomach and intestines.

**Prevalence.**—The disease affects buffaloes, and cattle naturally. It principally attacks buffaloes and is less common in cattle; horses are rarely if ever affected. It is most commonly met with in young animals, though older animals are not immune. Buffaloes, oxen, horses, sheep, goats, pigs, camels, rabbits, guinea-pigs, rats, mice and pigeons are all susceptible to inoculation, though camels, sheep and goats are very resistant to the disease. Rabbits are most suceptible. Dogs, fowls and men are immune.

It is most prevalent in low-lying lands subject to periodical inundation, as in the large river districts of the Punjab, where it occasions a heavy annual loss amongst cattle and buffaloes. Outbreaks usually occur during and after the monsoon season, or immediately following the Christmas rains. Almost any shower during the winter months will give rise to a few cases in affected districts.

**Bacteriology and infection.**—The causal agent is a short, thick ovoid bacillus of the pasteurella group. It is about 2 $\mu$ long, and ·4 to ·5 $\mu$ broad, but varies greatly in size and shape according to environment. It is found in every organ and fluid of the affected animal. In the blood, it is found singly or in pairs, each bacillus being surrounded by a mucoid layer. It is non-motile and stains very readily with all aniline dyes, the staining showing at the poles, with the centre clear. Loeffler's alkaline methylene blue or a one per cent. aqueous solution of methylene blue is suitable, but the bipolar staining is best brought out with a weak warm solution of carbolfuchsin or dilute Leishman.

According to laboratory experiments, the bacillus has only feeble powers of resistance outside the animal body. Ordinary disinfectants destroy it immediately. It is easily killed by drying. Direct sunlight destroys it in half an hour, and a temperature of 158° F. kills it in ten minutes.

Little is known of its existence outside the body. It is reputed to be a saprophyte, is probably ubiquitous, and possibly has greater powers of resistance than laboratory experiments appear to indicate.

Introduced into the animal body it grows with great rapidity, producing toxins of a very virulent nature. Enormous numbers of bacilli are discharged from the bodies of infected animals in the urine.

**Infection.**—Our knowledge of the mode of infection, and indeed of the whole subject of the disease, is very incomplete. Hitherto the disease has been variously supposed to bear relation to soil, water, and contamination of grass, infection thus resulting from ingestion; but recent experiments, in which young buffaloes have been fed with enormous quantities of virulent culture of bacilli without producing the disease, disprove an ingestion theory. On the other hand, inoculation readily produces the disease, and it is very probable that this is the real or only mode of infection. How it is brought about on the occasion of outbreaks of the disease is not exactly known. It may result from ordinary wound infection by soil or by ingestion if there are wounds in the mucous membrane of the mouth, but it is much more likely that inoculation is performed by biting insects. The periods of prevalence, *i.e.*, after rain, when biting flies are usually hatched out, rather favour this theory. The source from which they obtain the virus, and whether there is a "carrier" or not, are subjects that up to date have received no consideration. Until we are certain of the correct mode of infection, preventive measures can only be imperfectly applied.

**Symptoms and diagnosis.**—Often the first indication of the disease is an animal found dead. Suddenness of attack and death in a few hours should be particularly noted as characteristic features.

The following are the symptoms usually seen in the buffalo from natural infection:—Temperature 107° F. to 110° F., great depression; a painful, hot, hard swelling appears in the dewlap, throat, and between the lower maxillæ; swelling varies in size, but is usually large, does not pit on pressure, and is characteristic of the disease; there is dripping of saliva and a thick mucous discharge from the nose; the mucous membranes assume a hæmorrhagic deep red colour; the pulse is frequent; respiration is difficult bordering on suffocation, the nostrils being dilated and the chest heaving. Colic and intestinal symptoms as evidenced by diarrhœa and dysentery accompanied by severe staining, supervene. The animal lies down from sheer weakness, turns its head round to its flank, and dies within twenty-four hours.

**Post-mortem examination** shows a sero-gelatinous exudation under the skin, usually clear and serum-like, but sometimes of a hæmorrhagic appearance. The fourth stomach and intestines are much inflamed, the mucous membrane being brownish red in colour with hæmorrhagic patches throughout, and in places broken down and showing large raw ulcerations; the contents are bloody, and seldom is any food seen in them.

The mesenteric glands are enlarged, and there is an excess of peritoneal fluid.

The spleen, and blood as a whole, are of normal appearance. The bladder is inflamed, and the urine darker coloured than normal.

Endocarditis is almost a diagnostic *post-mortem* sign; myocarditis is generally found, and there is also an excess of pericardial fluid.

The lungs are generally normal or slightly œdematous.

A disease similating Hæmorrhagic Septicæmia has been met with amongst young mules of the young stock remount depots in India, cases occurring after the winter rains, and recurring in July and August. The symptoms were:—Temperature up to 105° F., dull and depressed appearance; conjunctiva deeply congested; respiration very accelerated; pulse very weak, serous and blood-tinged discharge from the nostrils; abdominal pain; collapse and rapid death. The *post-mortem* appearances were:—Serous membranes (peritoneum and pleura) studded with intense echymosis; the mucous membrane of large intestines inflamed, particularly at the pelvic flexure, with extravasation of blood into the lumen.

**Differential Diagnosis.—**

From **Anthrax** it is distinguished by the absence of the square-ended Bacillus Anthracis the absence of splenic enlargement, and the normal colour of the blood as a whole.

From **Blackquarter** it is readily distinguished by the absence of the the cold, painless, emphysematous or cracking swellings of that disease.

From **Rinderpest** it is distinguished by the history of its advent, the presence of œdematous swellings at the throat and dewlap, and the absence of mouth lesions.

A thoracic form of the disease may be mistaken for Bovine Pleuro-pneumonia Contagiosa, but this form does not appear to be common in India.

It has been the experience in the Military Veterinary Laboratory that organisms are often very difficult to demonstrate in the peripheral circulation, and are more easily demonstrated in fluid from the swellings. After death films are best taken from the heart blood.

Confirmation of diagnosis may also be resorted to in the inoculation of a rabbit, in which animal death invariably quickly results, a hæmorrhagic tracheitis being a diagnostic lesion.

## How to deal with an outbreak.

An outbreak is usually over in ten days, and the mortality is from 80 to 100 per cent. of those attacked.

Its disappearance is often as mysterious as its advent.

1. Completely incinerate the carcases of all animals dead from the disease.

2. If the condition of the animal permits, remove into isolation as soon as possible. If the animal is hopeless for treatment, or in a moribund condition, remove it out of harm's way by destruction, and incinerate carcase.

3. Isolate immediate incontacts, *i.e.*, those occupying standings on either side of the diseased, or all animals occupying the same fold, as in dairy farms.

4. Vacate affected folds as soon as possible. This is very essential. Change location after every fresh case.

5. Thoroughly disinfect all affected standings and folds, in accordance with " Routine of Disinfection " under the heading of " General Measures." Pay particular attention to the ground or floors, bearing in mind that urine and fæces are very infective. Burn all dung, bedding, broken fodder, etc. Do not omit mangers, feeding utensils, walls or anything likely to have suffered contamination.

6. Immunization: Hæmorrhagic Septicæmia serum can be injected to confer a passive immunity during an outbreak. The period of immunity conferred is short and consequently if animals are subjected to infection for a prolonged period they should be re-injected at intervals of two weeks.

A vaccine prepared by the I. V. R. I., Mukteswar, can be used to confer an active immunity for a limited period of about 2—4 months in districts where there is a seasonal incidence of the disease. The vaccine is sterile and there is therefore no danger of giving animals the disease and spreading infection.

D'Herelle in Indo-China successfully immunized cattle with Bacteriophage isolated from the fæces of recovered buffaloes.

7. Treatment. The disease usually runs so rapid a course that treatment is either of no avail, or impossible. If taken in hand early, antiseptic medicines such as carbolic or salicylic acids may be given internally. Enemas of warm water medicated with carbolic acid may be given every half hour. Injections of a 10 per cent. solution of carbolic acid into the swelling are recommended. " Anti-serum should not be given as it has no curative value." Diffusible stimulants are also indicated. Thin gruel with a little salt in it should be given to the animal to drink.

8. Certain pastures at certain times of the year are known to produce this disease: they should be avoided at these times. Lands in the vicinity of location of animals should be drained.

9. An outbreak may be considered over ten days after the last case, but affected folds should remain unoccupied for one month.

NOTES.

NOTES.

**Synonyms.**—Epizootic Catarrh; "pinkeye"; Stable-fever.

**Nature.**—Influenza is a rapidly spreading, contagious, febrile disease of a specially low or a dynamic type, chiefly affecting horses, and characterised by great prostration of strength, with a tendency to localisation in the mucous membrane of the respiratory and gastro-intestinal tracts, in the eyes, subcutaneous connective tissue, joints, or nervous system.

**Bacteriology and Infection.**—Various organisms have from time to time been asserted to be the cause of equine influenza including an organism of the pasteurella group, but none of them have been definitely proved to be the cause. It is now generally accepted that equine influenza is probably caused by a filtrable virus contained in the blood and other body fluids. Our knowledge, however, of the microbial cause of the disease is very imperfect. Secondary organisms no doubt play their part in the local manifestations of the disease.

Infection is conveyed by actual contact from animal to animal, by contaminated food or water, or by manure. It is usually introduced into a unit or a stable by a newly purchased horse. It is a disease of cities more than the country. Dealers' stables are often never free from it. Young animals are more prone to it than older ones, though when the outbreak is severe, animals of all ages are attacked.

Incubation is usually from one to three days.

**Symptoms and Diagnosis.**—Suddenness of the attack; large numbers attacked in rapid succession; great nervous prostration; high temperature; swelling of the eyelids and discharge of tears from the eyes; brownish red coloration of the conjunctiva and other visible mucous membranes; catarrhal symptoms of nose and throat, the discharge from the nostrils being first serious and later muco-purulent; digestive organs disturbed and the urine scanty and high coloured, are the common symptoms.

The disease is liable to show special predilection, for a given set of organs so that in different outbreaks different forms are presented, *viz.*, (*a*) a catarrhal form; (*b*) a thoracic form in which there is pneumonia, pleurisy and pericarditis in varying degree,—the pneumonia usually developing on the lower border of the lungs; (*c*) an abdominal form with flatulence, constipation, transient colic and diarrhœa; (*d*) a nervous form with extreme weakness, profound stupor and staggering gait; (*e*) a form where disorders of the eyes are very constant (pinkeye), the flow of tears being very profuse, and the conjunctiva bulging out between the eyelids; (*f*) an epizootic cellulitus form with dropsical effusions, particularly in the legs and on under surface of chest and abdomen; (*h*) and a rheumatoid form affecting muscles and joints. All these forms may, however, appear in different subjects of the same outbreak.

In India the disease usually assumes a mild form. As soon as it makes its appearance in a unit hard work should be stopped. All animals, except those suffering from fever or inappetance, should however be kept at healthy exercise. Early detection of a case is essential to avoid the complications which almost invariably result from working an animal when in the initial stage of the disease.

When symptoms subside work should be resumed gradually.

### How to deal with an outbreak.

A. (1) Strict isolation; cases to be removed from lines with the utmost despatch. Establish a sick camp a mile away and do not admit animals to the veterinary hospital unless it is set apart or converted temporarily for influenza cases only. Cases do well in the open, under trees.

The recording of temperatures of all animals in the unit twice daily should be instituted at once. The morning temperature should be taken before the animals go to exercise and any animal showing temperature of 101° or over given complete rest.

Only slow exercise should be given to animals during an outbreak even if the temperatures are normal.

(2) Separate attendants for the sick. They should be isolated too.

(3) Burn all manure during an outbreak and the manure of affected cases for one month after recovery.

(4) If the sick camp becomes foul, as it may do from the number under treatment, change after three weeks. This is especially necessary if the system of liberty paddocks is adopted.

(5) Disinfect in accordance with routine laid down under General Measures, paying particular attention to mangers, walls and pillars in front of animals,—buckets or watering and feeding utensils, water troughs.

The disinfection of the clothing of sick after recovery should not be forgotten.

B. It is seldom that isolation measures succeed in stopping the spread of the disease within a unit and as soon as fresh cases occur among the unit animals which are not isolated, the general policy of "mixing" should be adopted and every endeavour made to make the disease spread quickly within the unit. If his method is adopted all susceptible animals develop the disease within 14 days. By this means the duration of the outbreak is considerably shortened. While the disease is allowed to spread within a unit, endeavours should be made to protect other units in the station from infection. If, however, they become infected in spite of isolation measures the whole station should be treated as infected and all animals exposed to infection as soon as possible with a view to hastening the end of the outbreak.

Three weeks after the last case has been cured the station may be declared free from the disease.

Dealt with in this way the usual mild outbreak is usually at an end in six weeks to 2 months.

**Treatment.**—Influenza runs a natural course tending to recovery. Medicines are not of such importance as dietetic and hygienic care. Rest is a primary consideration. Fresh air is essential. Shade is necessary on account of nervous prostration and eye symptoms. The patient should be warmly clothed and the diet laxative. Plenty of cold water should be allowed, with occasional doses of nitrate of potash. Complicated cases should be treated as symptoms indicate. Care should be exercised with regard to purgatives. Strong purgatives should never be given. Constipation can be relieved with four ounce doses of sulphate of magnesia night and morning or a dose of linseed oil. Very little benefit is derived from antipyretics. Where there is entire loss of appetite, milk should be given and the patient tempted with lucerne, carrots, scalded, oats, etc. Good nursing is by far the best treatment. Rejected food should be destroyed, and the place where the animal stands disinfected daily.

The intravenous injection of 3 grammes of Neosalvarsan in 60 ccs. of distilled water has given excellent results in the medicinal treatment of severe cases.

Horses which have suffered from influenza do not recover their strength rapidly, and on no account should they be worked until they are in fit condition. Roaring is a sequel to the disease especially if worked too early.

**Prevention.**—As the disease is usually introduced into units by newly purchased animals from towns or places where the disease prevails, it is very necessary that all new arrivals should undergo a month's segregation before joining a unit. The disease being so rapidly infectious, rendering so many animals ineffective in a short time, segregation measures cannot be too strictly enforced.

The chlorination of drinking water or the addition of permanganate of potassium has appeared to check the spread of the disease in some outbreaks. It may be tried on non-affected units in a station during an outbreak.

A vaccine or serum for protective inoculation would prove a great boon, but a suitable one has not yet been produced.

Poles of Rotterdam gave 5 ccs. of virulent blood to 400 Remounts and then turned them out loose in kraals giving no treatment. 90 per cent. developed symptoms in varying degrees but all recovered with no complications.

NOTES.

**Synonyms.**—Jhoolak. Zohri (Sudan).

**Nature of Disease.**—Jhooling is a contagious disease of camels affecting the skin and subcutaneous tissue, characterised by the formation of tumours of a fibrous nature, which break down and suppurate, terminating in raw ulcerous patches.

**Susceptibility.**—So far as is known the disease is peculiar to the camel.

Healthy camels kept in contact with affected ones readily contract the disease.

The disease can be transmitted to healthy camels by rubbing pieces of jhooling lesions on the scarified skin. It is not easy to transmit it unless the skin is first scarified.

**Prevalence.**—It is common disease in camels in India, and is widely distributed throughout the Punjab. It occurs more commonly in the cold weather.

**Bacteriology and Infection.**—The causal organism has not yet been demonstrated. Examination of the pus reveals a pure culture of streptococci. These organisms are found in large and numerous masses in the upper layers of the inflamed tissue. The histology of the lesion is that of a rapidly developing, acute pyogenic infection. The natural method of infection is probably by inoculation only.

**Symptoms and diagnosis.**—The first symptom is a hot, hard, painful swelling, varying from 1 to 5 inches in diameter, and may appear on almost any part of the body, but the commonest sites are the neck, hind quarters and belly. After a few days the swelling becomes irritating, and the camel will, if possible, gnaw it, producing a raw patch. Sooner or later suppuration occurs, to be followed by healing. The length of time it takes for a lesion to heal varies considerably, depending on its size and depth. The site of old lesions is marked by a white patch which lasts for several months. Lesions are usually multiple, and the disease may occasion loss of condition. Some lesions are very intractable, and if situated anywhere under the palan or girths may throw a camel out of work for a considerable time. Diagnosis is based on the clinical picture of the case. The disease appears to be strictly local, and no lesions have been reported affecting the internal organs.

**How to deal with an outbreak.**

With regard to the treatment there is no specific remedy. The lesions must be treated according to their condition. Excision is to be recommended, followed by dressings as for an open wound. Dressings at first require to be stimulating. In obstinate cases changes of dressing are indicated. The following treatment is recommended by Cross:—

A strong red iodide of mercury blister should be applied, and after 3 days should be washed off with soap and water. The diseased area

should then be excised and finely powdered permanganate of potash applied. The permanganate of potash must be well rubbed in and not simply dusted on. Three dressings at intervals of 4 days are usually sufficient.

Another treatment recommended is excision of the lesion and the application of pure phenyle or carbolic acid. The following day the wounds should be thoroughly washed with water and then treated with Blackwash (30 grains calomel, half an ounce of glycerine, $1\frac{1}{4}$ ounces of tragacanth mucilage and lime water to make 10 ounces of lotion).

The application of heated ox bile to lesions assists healing and has been found to give better results than the older methods of treatment (*e.g.*, permanganate of potash crystals). In unexcised lesions the sloughing process is slower but when the scab comes off the ulcer is cleaner and heals more rapidly, while heated ox bile applied, to excised lesions hastens healing and the separation of adherent necrotic tissue. A fly deterrent dressing should be applied around the lesions.

## Preventive Treatment.

The contagious nature of the disease is well recognised by camel-men, and it is the custom, if a camel is suffering from lesions on the neck, to place it first in the string, whereas if lesions are about the hind-quarters the camel is placed last.

The addition of four to five ounces of salt to the daily ration has been found by Peck in Somaliland to prevent the development of lesions and to hasten the healing of existing lesions. As a result of trials in India this amount of salt is now given to camels in military employ.

An affected camel should be immediately isolated, and a thorough inspection of the remaining camels of the unit should be made immediately. This inspection of healthy camels should be carried out daily, and any suspicious cases immediately brought to notice. Thorough measures of disinfection should be carried out according to routine. Incontacts should be isolated separately from the affected. Special attention should be paid to the disinfection of the standings, line gear, palans, etc. Contaminated fodder should be burned. The isolation of affected camels with their attendants must be strict. The dressing of affected camels must be carried out under strict supervision to ensure that contaminated dressings are properly disposed of. Dressers should wash and disinfect their hands thoroughly after attending to Jhooling cases, and must be careful to avoid contamination of their clothing or boots which must be regularly washed. All newly purchased animals must be carefully inspected before admission to unit lines.

# NOTES.

# NOTES.

**Synonyms.**—Chronic bacterial enteritis of cattle.

Chronic pseudo-tuberculous enteritis of cattle.

**Nature of disease.**—Johne's disease is a chronic enteritis affecting particularly cattle, but is known to affect also sheep, deer and goats. The causal organism is a small acid-fast bacillus, resembling in some respects the tubercle bacillus.

**Prevalence.**—The disease has a world-wide distribution. In recent years it has been demonstrated to be widely distributed throughout India, and outbreaks have been dealt with in Dairy Farms and Remount Depots situated in the North and South of India. In Great Britain the disease is very prevalent, and is looked upon as second only in importance to Tuberculosis.

**Bacteriology and Infection.**—The disease was first recognised as one of a peculiar types by Johne and Frothingham in 1895. The bacillus is rod-shaped, 1 to 1·5 μ long and ·5μ wide. These are regular cylindrical, slightly curved, and rod-shaped forms. It has the same staining properties as the tubercle bacillus, being acid fast. It is a difficult microbe to cultivate artificially, and will not grow on ordinary culture media. Twort and Ingram first devised a suitable medium. They found that by cultivating and killing the Timothy grass bacillus (B. Phlei) and mixing the result with the nutrient media, the bacillus of Johne grew most successfully.

Infection takes place by ingestion of foods contaminated by excreta of infected animals. The habitat of the bacilli being the intestinal mucosa, they are voided in large numbers with the fæces, giving rise to heavy soil infection.

**Symptoms.**—The disease is very slow in its development. Affected animals may retain their condition for a long time and shew no clinical symptoms or may never shew symptoms at all. For this reason the disease is very insidious and many animals may have become infected before it is discovered.

The first of the clinical symptoms to be observed is loss of condition and unthriftiness which may be followed by progressive emaciation. In milch cows there is a diminution of the milk yield. The temperature varies from normal to 2 or 3 degrees of fever. As the disease progresses, diarrhœa of a profuse and foetid nature sets in and the presence of bubbles in the excreta is a marked feature. An animal which once shews these advanced symptoms rarely recovers. Any debilitating influence such as the contraction of another disease, or parturition, may intensify the progress of Johne's Disease and lead to the development of acute symptoms in a latent case, resulting in death.

**Diagnosis.**—One may be able to make a fairly accurate diagnosis from the history of the outbreak and the clinical picture presented. It would be very rare to find only one visibly affected animal in a

herd. It may be possible to make a positive diagnosis by pinching off a small particle of the mucous membrane of the rectum, and examining microscopically for the bacilli. It may also be possible to demonstrate the bacilli in fæces previously treated with antiformin. A negative result, however, does not justify a diagnosis of "not Johne's Disease," since the organism are not always easy to find. Several diagnostic agents of the nature of tuberculin for tuberculosis have been tried. One preparation known as Johnin is prepared from the specific bacilli, but although a temperature reaction may be given in a certain number of cases, it cannot be said to be a reliable test.

Another diagnostic agent is Avian Tuberculin which may be injected subcutaneously or intradermally. The former is more reliable. The animal should first be tested with bovin tuberculin for tuberculosis since if affected with this latter disease a reaction would be obtained to the Avian Tuberculin. Major G. W. Dunkin, Field Research Laboratories, Mill Hill, has produced a diagnostic agent which he reports as encouraging. It is an agent prepared in much the same way as tuberculin and which he describes as "Tepsin". He uses it as a double intra-dermal test in the same way as one uses concentrated tuberculin when adopting the double intra-dermal test for tuberculosis. The test can be carried out simultaneously with the tuberculin test, one test being done on the right side of the neck and the other on the left side.

Diagnosis may be confirmed by *post-mortem* examination. The disease is a chronic entiritis affecting both small and large intestines. The small intestines and ileum in particular are principally affected. In advanced cases the bowel wall will be seen to be thickened and the mucous membrane shews a characteristic wrinkled or corrugated appearance, most noticeable in the last part of the ileum. The thickening of the mucous membrane is less marked in the large intestine. There is no congestion or ulceration. The mesentric glands are enlarged and œdematous. Organisms are found in abundance in scrapings from the affected parts of the bowel.

## How to deal with an outbreak.

Medicinal treatment is of little avail. One can only treat the disease symptomatically. Diarrhœa may be checked by opiates and astringents. Tonics may be tried and good feeding is essential.

Animals which are only mildly affected, and shew no clinical symptoms may be fattened for the butcher, or in the case of working bullocks may be worked in isolation. Recovery may take place in such cases, but as soon as an animal shews marked symptoms of the disease, slaughter is to be recommended.

**Preventive measures.**—Immediate segregation of all affected and suspected animals. Testing of the whole herd dividing it up into

affected, suspected and healthy. Destroy the severely affected, and keep the mildly affected in isolation. The test should be repeated periodically. Special attention should be given to the disposal of excreta of affected animals which is highly infective. On affected premises calves should be reared separately. As sheep and goats are susceptible these must receive attention in dealing with an outbreak. Pastures which have been used by affected animals should be vacated and put under the plough.

# NOTES.

## MANGE

**Synonyms.**—Scabies, scab, itch, acariasis.

**Nature.**—Scabies is a very contagious skin disease, due to **acari or** animal parasites of the arachnida or spider family. It is met with in all animals and men.

**Prevalence.**—In India, horses, mules, donkeys, and cattle in military service, are rarely affected. It is very prevalent amongst camels.

Prevalence bears relationship to neglect and filthy surroundings; and it is often the accompaniment of debilitating diseases in camels.

**It is not common in the stabled, groomed and well-cared for animals in military employ.**

It is a great scourge in times of active service, causing an infinite amount of trouble and loss.

### Parasitology and infection.

1. **Varieties of acari causing mange:—**

There are four genera, *viz.*—

(*a*) Sarcoptes.

(*b*) Psoroptes.

(*c*) Symbiotes or Chorioptes.

(*d*) Demodex.

2. **Varieties affecting animals in military service:—**

*In the horse and mule.*—

(*a*) Sarcoptic—the most difficult to cure.

(*b*) Psoroptic—the most common.

(*c*) Symbiotic or Chorioptic—infrequent, and comparatively harmless.

(*d*) Demodex—comparatively harmless.

*In camels*:—

Sarcoptic—common.

*In cattle*:—

Psoroptic—rare.

For general information in connection with other animals, it may be mentioned that the mange or scab in sheep, which gives rise to much loss in some countries, is psoroptic; sarcoptic mange in sheep is limited exclusively to the head, avoiding places covered with wool. Pigs are not commonly affected, and the variety is Sarcoptic. In dogs the ordinary scabies are sarcoptic; they also suffer from Follicular mange caused by another genus of the arachnida, the Demodex Folliculorum (the "black head" in human beings). The disease in cats is sarcoptic. In both dogs and cats a Symbiotic form is met with in the auditory canal of the ear (Symbiotic otacariasias).

3. **Morphology and how to tell one variety from another.**—For the intelligent treatment of cases, it is very important to be able to distinguish one kind from another, particularly sarcoptic from psoropotic.

Morphologically all psoric acari are round or oval whitish looking parasites, about ·2 to ·8 millimetres (1/25 to 1/32 of an inch) in size, without any differentiation of head, thorax and abdomen. They possess four pairs of legs, two pairs in front and two pairs behind, terminating in long bristles or ambulatory suckers. Mouth parts are well marked. They have no eyes: respiration is cutaneous. The sexes are distinct: the females are larger than the males, and they exist in greater numbers (in sarcoptic mange the males are in the proportion of 5 or 6 per cent. to the total).

The Sarcoptes [size ·2 to ·5 mm.] are **round** or oval, have a short strong rostrum with two cheek pieces, short thick legs, **the hind pairs not visible when viewed from the dorsal aspect,** the ambulatory suckers are on long single pedicles. These parasites can only be seen by the microscope (low power).

The Psoroptes [·5 to ·8 mm.] are larger than the Sarcoptes, they are oval in shape, the rostrum is long and has no cheek pieces, the **legs are long and are all visible from the dorsal aspect,** and have ambulatory suckers on long three jointed pedicles. The parasites can be seen with the naked eye or an ordinary magnifying glass by placing scabs on a piece of black paper.

The Symbiotes or Chorioptes [·3 to ·5 mm.] are oval, with rostrum as broad as it is long, all legs are visible from the dorsal surface and have wide suckers on short stalks.

The Psoroptes and Symbiotes are readily found; the Sarcoptes and Demodex are more difficult.

4. **Life History of the Parasite.**—Under favourable conditions they multiply very rapidly. The females are oviparous, producing during the course of their lives from twenty to twenty-four eggs ovoid in shape and with a transparent shell. Incubation of the eggs on the animal body only lasts a few days, from 24 hours to 7 days, depending on the season and the individual, each egg producing a hexapod larva, which by a process of moulting passes through a nymph stage, finally becoming a sexually mature parasite in fifteen days.

The duration of the germinative faculty of the egg outside the body is not known. It is certain that, stored up in blankets, the lining of harness and saddles, the wood work of stables, and even soil, it can retain its vitality for a considerable time like any other egg, and it is to this fact that outbreaks and recurrence of outbreaks are usually due. It has even been considered that animals themselves may in some unaccountable way store up the eggs, or harbour the parasite in some stage of its development, to reproduce the disease in them after a few month's apparent cure.

The Sarcoptic fecundated females burrow into the skin forming intra-epidermic galleries where the eggs are laid. Observed in man (owing to pigmented skin they cannot be seen in horses) these are from 1/5 to 4/5 of an inch long, and are rapidly formed in about 15 to 30 minutes. This habit explains the difficulty in effecting a

cure in this form of mange, and may account for the recurrence of the disease after a lapse of time in apparently cured animals.

The Psoroptes live on the surface of the skin, piercing it to obtain juices. They are active travellers.

The Symbiotes or Chorioptes also live on the surface, feeding on epidermic scales, and have no tendency to migrate from their usual habitat, *viz.*, legs of horses, or the root of the tail in the ox.

5. **Infection.**—A dirty skin, debility, neglect of grooming, want of of general care, and insufficient food are all predisposing factors of the disease, particularly in the psoroptic variety. An idle horse will contract the disease sooner than a working horse for the reason that his coat is dirtier. Even clean horses, however, offer no resistance to the Sarcoptic form.

Infection is by immediate contact, or through the medium of blankets, saddles, harness, grooming utensils, bedding, stalls, partitions, fences, rubbing posts, etc.

The Psoroptic variety is very rapidly spread when animals are huddled together, as in railway trucks, or on board ship, or in flocks as in the case of sheep. Bedding is greatly responsible for spread.

Sarcoptic mange is slower in infection by reason of the life history of the parasite.

Each species of animal has its own mange, and for the most part, the disease of one is not communicable to the other, or the parasite will not live for any length of time except on its particular host. Thus the human itch parasite (sarcoptic) only lives temporarily on the horse and the horse sarcopt, although transmissible to man, dies spontaneously in 15 days to 6 weeks, and yields to simple treatment. The horse sarcopt is not transmissible to cattle. Sarcoptic mange in camelmen (sarwans), contracted from their camels is, however, quite common. Instances, too, are on record of the transmission of sarcoptic mange to human beings and horses from dogs and cats; but outbreaks in horses have not been attributable to either of the latter animals. The burrowing Sarcopt in any species should never be trusted.

Psoroptic mange is not inter-communicable, and the same applies to Symbiotic mange.

**Symptoms and Diagnosis.**—The symptoms common to all animals and all varieties are an intense itching, and a depilated condition of the skin in different regions affected. The itching is due to an irritant matter deposited by the parasite, and is most intense in the psoroptic form. It is greater at night than during the day, in warm weather than in cold, in the stable than out of it, and when clothed than unclothed. Animals evince great pleasure on being scratched. Positive diagnosis is the finding of the parasite. The different forms more or less select particular parts of the body.

In horses the sarcoptic form usually commences on the upper part of the body or neck, most commonly on the withers, and spreading in bad cases all over the body excepting the legs. On examination

of the skin, particularly at the margin of an affected part, minute points or papules will be seen and felt. Hairs fall out, small crusts are formed, and by dissemination large dry patches result. Eventually the skin becomes thickened, wrinkled, and covered with a dry powdery crust.

To detect the parasite, which is not easy in this form, scrape the skin at the margin of an affected part, where youngest papules exist, until blood shows, after having placed the animal in the sun for an hour to increase the activity of the parasite. If Mange scrapings are sent to a Laboratory in an envelope, the latter should be so packed as not to permit of the escape of the parasites. Place the scrapings in a test-tube with 10 per cent. caustic potash or soda, and boil for five minutes over a Bunsen flame. The mixture is then centrifuged for a few minutes to throw down all solid particles, the supernatant fluid is poured off, and the residue after mixing with a little glycerine is smeared on slides for examination.

Another method of treating the residue (known as the sugar flotation method) is to half fill the test tube with clean water and then fill up with sugar solution (1 lb. sugar to ¾ pint of water). The tube is then centrifuged. Lower a round cover slip on to the surface of the liquid in the test tube (for this purpose the cover slip may be attached to a pencil by means of a small piece of plasticine), touch the surface of the liquid and withdraw. The cover slip is then mounted in the ordinary way. The specific gravity of the sugar solution causes the parasites to rise to the surface whereas the debris sinks.

*The Demodex.*—The body is wormlike in outline the abdomen being elongated. There are four pairs of short legs each with three segments. There are no suckers, no eyes, no anus and no sexual differences are apparent. The parasites are found in the hair follicles and sebaceous glands.

Psoroptic mange may commence on any portion of the body excepting the legs, but usually has a liking for the upper part of the neck at the root of the mane and the tail. The pimples that are caused by the bite of the parasite discharge a serous fluid, and the crusts that are formed remain moist and viscid, unlike the dry crusts of the Sarcoptic variety. The parasites are easily detected by a magnifying glass by placing crusts on a piece of black paper and exposing to the sun. Their numbers and movements make diagnosis easy.

Symbiotic mange is limited to the legs in horses, and practically affects hairy heeled horses only. It is indicated by the animal stamping, rubbing one leg against another, even to causing bleeding with the shoe. The disease follows a very slow course. The parasite is easily detected.

*Demodectic Mange.*—In the early stage the parasites invade the hair follicles. Inflammatory reaction results followed by a stage of suppuration and a pustular eruption. Affected areas are generally the neck, withers and croup. Well conditioned animals seem more

liable to infection than others. There is no direct proof that the disease is contagious.

Mange may at first sight be mistaken for phthiriasis (lousiness), but the broken hair without actual depilation and the presence of lice easily differentiates the latter.

Certain Eczemas, particularly in dogs, and perhaps succeeding strangles and pasteurella diseases in young horses, are confusing; and it not infrequently happens that an old case of so-called Eczema, which has been under treatment in hospital for such and returned to troop lines after apparent cure, breaks out again concurrently with the appearance and spread of Sarcoptic mange amongst other animals of the unit. Usually, there is less pruritis in Eczema, and other clinical signs permit of differentiation; but the absence of the parasite, its eggs, fæces or portions of its body is the real distinguishing feature.

Horses stabled in proximity to fowl-yards are sometimes attacked by Dermanyssus of fowls and pigeons, and the condition resembles mange. On removal of the animals the condition ceases; depilation is also never the same as in mange.

On board ship, horses on the upper decks are apt to lose the hair of their back and quarters from the action of the spray. This is mentioned, but ought not to be mistaken for mange.

**Mange in Camels.**—This is sarcoptic, but the parasite is more easily found than in horses. It is larger, and can be detected with the aid of a magnifying glass.

The symptoms vary little from those in the horse, excepting that it usually begins on the inside of the thigh, scrotal region and flank and in bad cases may extend all over the body. It is often the accompaniment of a debilitating disease such as surra, and in such cases progresses rapidly, animals loosing flesh. Pruritis is violent, the animal rubbing its legs together, scratching, and rubbing against trees, the ground and frequently its companions.

From want of succulent food, camels may suffer from an Eczema with loss of hair, but the depilation in such cases commences on shoulders, neck and hump, and no parasites are to be found.

**Mange in Oxen** is not frequent. When it occurs, it is the psoroptic form, commences at the upper part of neck and withers (hump) and is very amenable to treatment.

Care should be taken not to confuse the depilation of hair, which occurs at the back, dewlap and shoulders in the spring from change of coat, with mange.

A symbiotic form is sometimes seen at the root of the tail; it is localised in that region, and easily cured. It is of no importance whatever.

### How to deal with an outbreak.

The disease being so insidious, difficult to cure, at least in the Sarcoptic form, and liable to recur, *half* measures are of no use;

a systematic and radical **method must be adopted and personally carried out** by the officer charged with the responsibility of dealing with the outbreak.

1. Strictly isolate all affected animals, and also doubtful cases.

2. Closely examine all animals of a unit for signs of the disease as soon as possible; make daily inspections.

3. Group animals into (*a*) affected, (*b*) doubtful (*c*) free,—transferring them to each group as occasion requires. Doubtful cases should include immediate incontacts in a stable, animals that have been groomed by the same attendant or by the same grooming utensils as an affected animal, or which in any way can be shewn to have been directly exposed to the contagion as by bedding, interchange of contaminated clothing, saddlery, harness, grooming, kit, etc.

4. Hold an enquiry into the above and take action accordingly.

5. Vacate the stabling or standings and institute a thorough process of disinfection.

6. Clip all animals from an infected stable, carefully burning the hair clipped off, and placing the clipping machines in paraffin oil after each case. If the disease has appeared in several stables or lines, clip the whole unit.

7. Burn all bedding contaminated or likely to have become contaminated.

8. Abolish straw or grass bedding throughout the unit: use sand, and let it remain down, removing soiled portions and droppings.

9. Fire the surface of standings stables walls, partitions, pillars, and mangers where affected and doubtful animals have stood, thoroughly saturating with solution of Chloride of Lime or Carbolic acid afterwards. The process should be repeated.

10. Burn the clothing of all affected animals, and do not issue fresh clothing to them until three months after cure.

11. Take off the clothing of doubtful animals, disinfect it as laid down in "Routine of disinfection" and do not re-issue until certain that animals are not affected, allowing a clear month to elapse.

12. Collect all grooming kit of (*a*) affected, (*b*) doubtful, and deal with it as laid down in "Routine of disinfection" under "General measures". Do not re-issue to affected cases until after cure. The washing received in treatment takes the place of grooming, and should the latter be required, perform it with a grass plat, which destroy immediately after use. After cure, brushes and curry-combs may be issued, but they should be stamped with a large M indicating mange, each animal having his own articles. These should be placed daily in disinfectant solution for one hour after use, a solution being maintained for that purpose.

In doubtful cases grooming articles should not be re-issued until it is certain that mange is not present. One clear month should be allowed, grooming being performed by grass plats during that time, each animal having its own wisp.

13. Collect all harness and saddlery of (*a*) affected, (*b*) doubtful. Disinfect as laid down under "Routine of disinfection". Without exception destroy the lining of collars and panels of saddles and palans that have been used on affected animals. Numdahs can be easily disinfected.

14. Forbid interchange of equipment in the unit during, and for three months after, the outbreak.

15. Disinfect separately at the earliest opportunity the clothing, harness, saddlery and grooming kit of unaffected animals (the free group) of the entire unit as a precautionary measure. For grooming utensils a bucket of disinfectant solution should be maintained in the stable, into which articles should be placed for an hour after use.

16. Use as little clothing as possible, consistent with warmth, in the free group. Whether all clothing should be abolished in a unit depends on the extent of infection, probable as well as actual. Experience has shown that often this is absolutely necessary.

17. **Treatment of affected and doubtful.**—*Caution*! Overtreatment and excessively irritating dressings must be avoided. The use of linseed oil as a basis of treatment, especially in hot climates, is not advisable. It tends to blister, especially in the sun, probably owing to other oils as adulterants, and, moreover, it forms a varnish on the hair and skin rather difficult to remove.

Begin with the doubtful (including incontacts or otherwise exposed). Clip off the hair from head to foot and burn it; singe, wash and scrub all over with soft soap and warm water; scrape and allow to dry. Then dress all over with some simple dressing such as phenyl solution or if there are any doubtful patches, dress with a mange dressing mentioned below. Immerse the clipping machine in paraffin oil after clipping each animal. Keep animals under careful observation.

Affected cases come next. Clip and singe as above mentioned, taking extra pains to burn the hair clipped off, and to disinfect the clipping machine. Smear all over with soft soap, rubbing it into affected parts to soften the scrabs. Leave it on for half an hour to an hour. Then wash and scrub into a lather with warm water until all scabs are removed. Apply more water to take away excess of soap, scrape and dry with wisps. Then apply a mange dressing.

There are many formulæ used, different people having their own particular fancy. To ensure safety and to catch all wandering parasites dress animals as a rule all over, certainly at the first dressing. Dressings having an oleaginous basis should not be applied over the whole surface of the body at one time, as the sudden arrest of cutaneous function causes harm to the individual. Half the body can be done at one time, and the other half 48 hours afterwards, any affected or doubtful places being dressed at the first time.

Oleaginous dressings are the best, and have the advantage of requiring less frequent applications. Dressings containing sulphur and tar are very good parasiticides.

A thorough dressing of paraffin oil one pint, soft soap 1 lb., and water one gallon is a most efficient remedy. Calcium sulphide [made by boiling together 2 lbs. of Sulphur, 1 lb. quicklime, and 2 gallons of water and stirring frequently until the ingredients are combined and diluted by adding three parts of water to one part of the mixture] is a good dressing, and one which can be used as an all-over dressing at one application. The solution should be applied warm (110°F.). Mange Hospitals in France during the Great War used this solution extensively. The solution was contained in concrete baths in which the cases were immersed.

Crude fuel oil is an excellent dressing, and two or three dressings usually suffice to cure. The oil softens the scales and crusts and when these are removed by gentle hand rubbing, the skin is found to be soft and pliable. It was used with success during the Great War in Mesopotamia and has since proved its value in this and other theatres of war.

Sulphuration, *i.e.*, the subjection of an animal to the action of sulphur dioxide gas has been largely practised. Special gas chambers have to be constructed for the purpose. A 3 per cent. to 6 per cent. concentration of gas is necessary with an exposure of about half an hour. The treatment is repeated on the 8th day and two such treatments are said to effect a cure. Preliminary dressing by hand of the ears, jowl, muzzle and other parts of the head not exposed to the action of the gas is necessary.

The following are very efficient remedies:—

Sulphur 2 oz., Creosote 1 oz., Lard 8 oz. (or oil instead of lard).

Sulphur 2 oz., Mercurial Ointment ½ oz., Lard 8 oz.

Sulphur 1 part, Tar, or Oil of Tar 1 part, 8 parts common oil.

Sulphur 2 parts, Oil of Tar 1 part, Pot. Carb. 1 part, Lard or Oil 10 or 12 parts.

Sulphur 1 part, Oil of Tar 1 part, Soft Soap and Lard each 2 parts.

Tar 1 or 2 parts, Vaseline, Lard, or Alcohol 10 parts.

Creosote 1 oz., Methylated Spirit 15 oz., Water 40 oz.

Sulphur and Oil made into a thin paste.

Taramera Oil either by itself or in thin pastes with Sulphur.

Cheer pine Oil and Sessamum Oil half and half with Sulphur.

The three latter are excellent remedies in camels.

Corrosive Sublimate 1 in 2,000 is a convenient remedy on active service.

All mange dressings should remain on the skin for several days, and be lightly rubbed in with the hand daily. After that they should be washed off with soap and water, and re-applied, affected parts more particularly being dressed. No object is gained in very frequent applications and over-dressing; as irritation of the skin is only produced, confusing the result. It is much better to leave the dressing on, occasionally rubbing it in.

Two or three dressings should effect a cure of the psoroptic form. Treatment should be extended over three weeks in the Sarcoptic form, and the case watched after that time.

After cure, a thorough washing and scrubbing must be given. The patient must have exercise to keep up the action of the skin, and a liberal amount of good food allowed.

Treatment is practically the same for all animals. The use of mercurial dressings in cattle requires care, owing to their habit of licking. Too frequent use of mercurial dressings may also cause mercurialism by absorption. An oily dressing over a large surface in sheep frequently causes death. Arsenical dressings (arsenic one-pound, carbonate of potash 1 lb., water 20 gallons) or non-poisonous dips such as McDougall's, Little's etc., are remedies in their case, care always being taken to allow animals to drip in a fold and not on their gazing ground after dipping.

18. **Working isolation.**—Units should be placed in working isolation until the cure of the last case. Cured cases should be segregated up to three months; but they may perform work by themselves after cure. After that time they may rejoin their unit.

### Mange on active service.

As the disease is so difficult to treat on active service, and gives such an infinite amount of trouble, the following important points should be noted:—

1. No animal, especially camels to proceed on service with the slightest signs of the disease.

2. Occurrence to be at once reported and frequent inspections made.

3. Affected to be sent to a Field Veterinary Hospital and dressed in isolation.

4. As so much bulky dressing is required for treatment, the latter is best carried out at the base or some convenient centre, to which animals should be sent after a preliminary dressing.

# NOTES.

## PARASITES OF THE ALIMENTARY TRACT OF HORSES.

### General classification of parasitic worms.

The Vermes are subdivided into the Platyhelminthes, Nemathelminthes and the Annelida.

1. **The Platyhelminthes.**—(Flat worms)—
   (*a*) **Trematodes**—the flukes.
   (*b*) **Cestodes**—the tapeworms.
2. **Nemathelminthes.**—Smooth-bodied round worms—
   (*a*) Nematodes—
   (*i*) Ascaridæ.
   (*ii*) Spiruridæ.
   (*iii*) Strongylidæ.
   (*iv*) Oxyuridæ.
   (*v*) Eustrongylidæ.
   (*vi*) Trichinellidæ.
   (*b*) Acanthocephala.
3. **Annelida.**—Segmented round worms.
   **Hirudinae.**—The leeches.

### Platyhelminthes.

(*a*) **Trematodes.**—The only fluke worms of importance in the horse are :—

The Gastrodiscus Aegyptiacus.

The Gastrodiscus Secundus.

These are very common in India. They inhabit the small intestine and are frequently found in the fæces of horses after the administration of a vermifuge. They do not appear to cause severe constitutional disturbance. In the fæces they appear as fleshy beans from ¾ to 1 inch in length and ½ inch in breadth.

(*b*) **Cestodes.**—Three species of tapeworms occur in equines, *viz.*, Anoplocephala perfoliata, Anoplocephala mamillana and Anoplocephala plicata.

Of these the first named only is common. These tapeworms are all 'unarmed' and when found are always free in the lumen of the bowel.

**Habitat.**—The small intestine, caecum and colon. Anoplocephala mamillana has also been found in the stomach.

**Symptoms.**—There is no evidence to shew that these parasites ever give rise to digestive or other disturbances in the host.

**Tapeworm larvae in equines.**—Hydatid is fairly common in horses in the British Isles, about 20 per cent. being affected. The condition,

however, is generally only detected on *post-mortem* examination. Larval forms of the following tapeworms have been met with in the horse :—

**In the cranium.**—Cœnurus cerebralis, the larval stage of Multiceps of the dog.

**In the peritoneum.**—Cysticercus tenuicollis, the larval stage of the Tænia Hydatigena of the dog.

**In the lungs and liver.**—Echinococcus granulosus, the larval stage of the Echinococcus granulosus of the dog and cat.

## Nemathelminthes.

### (a) Nematodes.

#### (i) Ascaridae.

Only one species inhabits the intestine of the horse, namely the Ascaris equorum (megalocephala).

**Life history of ascaridae in general.**—Recent work has shewn that the life history of all ascaridæ is essentially the same.

**Development outside the body.**—Eggs furnished with resistant shells are deposited unsegmented. Embryos are developed in about four weeks at a temperature of 25°C. They do not hatch out, outside the body.

**Development inside the body.**—Embryonated eggs ingested, hatch out in the pylorus or duodenum, and larvæ penetrate the intestinal wall, reaching the liver either by direct migration, or by being transported by the portal circulation. They are then carried to the capillaries of the lungs and escape into the air sacs, migrating up the finer bronchioles, bronchi, and trachea to the pharynx. At the pharynx they are swallowed, and again pass down to the small intestine. If the host is not the correct one, the larvæ pass to the exterior with the fæces; if it is the correct one, they develop to maturity in the intestine.

**Symptoms.**—Excessively heavy infestation will produce a condition of chronic pneumonia and unthriftiness, owing to the effect of the migrating parasites in the lungs. This condition is clinically recognisable in young pigs, and there is reason to believe that it occasionally occurs in foals also, especially when reared under very insanitary conditions, and in a confined space.

There would appear to be also some degree of toxæmia as a result of infection.

**Size of parasites.**—The female is 15-30 cm. long. The male is shorter. The worms are readily recognised by their large size and thickness.

**Characteristics of the ova.**—Ascarid eggs are oval with a thick shell. They are unsegmented in fresh fæces. They often have an external albuminous coating.

**Diagnosis.**—It is common for horses to pass odd worms from time to time; as a rule this cannot be regarded as an indication of severe infestation. Where heavy ascarid infection is suspected, a vermifuge test should be carried out or the fæces examined for the ascarid eggs in the manner hereafter described.

**Treatment.**—Oil of Chenopodium is one of the best medicinal agent to employ against Ascarids in the horse. The dose for horses is 2-4 drachms. The animal should be fasted twenty-four or thirty-six hours. The drug should be given as a single dose and never in small repeated doses. It should be mixed with, or followed immediately by $1\frac{1}{2}$ pints, or more, of linseed oil. Given in this manner no toxic effects result.

Carbon bi-sulphide is said to have an almost 100 per cent. efficiency for ascaridæ in horses. It should be administered in capsules or by the stomach tube the dose being 2-6 fl. drms. If desired, the drug may be administered in two doses of four drms. each, at two hours interval; or three doses of three drms. each, at hourly intervals. The repeated dosage is more efficient than the single dose, and it gives an opportunity of suspending treatment should the drug seem to have bad effects. A purge should not be given within at least several hours of the carbon-bi-sulphide.

Phenothiazine has recently been used and is efficacious. For method of use see page 174.

(ii) **Spiruridae.**

**Gastric Harbronemiasis.**—Much importance is attached to this infestation as a cause of debility amongst horses in India and Australia, the debility in some cases being accompanied by symptoms of incoordination. It has been stated that probably as many as 60 per cent. of all cases of debility among Australian horses in India are due to Harbronemiasis.

Three varieties of parasites are found, *viz.*, Habronema Megastoma, Habronema Microstoma, and Habronema Muscæ.

**H. Megastoma.**—Adult female up to 13 mm. long, male 7-10 mm., colour white. They infest the submucosa of the stomach, usually in the pyloric portion, and their presence may be recognised by oval or rounded prominences, varying in size from that of a hazel nut to a walnut. The mucous membrane covering the tumours is unaltered with the exception of a number of perforations at the summits, which communicate with the contained cavities. Within these cavities are lodged the worms which, on pressing the tumours are evacuated together with purulent material. In advanced cases the tumours have been known to attain the size of an orange or a cocoanut. The contents contain cheesy looking pus and nests of the H. Megastoma. Occasionally perforation of the stomach wall, with peritonitis and secondary splenic abscesses results.

**H. Microstoma and H. Muscae.**—Adult female about 12-27 mm. long, male 10-20 mm. They are thus about twice the size of the preceding. These worms are found lying free on the mucous surface of the

stomach, generally the cardiac portion. They do not penetrate the lining membrane. On *P. M.* examination it is not uncommon to find myriads of these worms present, and they may give the appearance of undulating movement to the stomach contents. If the stomach contents are emptied out and the surface washed, their presence is readily seen. Mere washing does not remove them; they require to be scraped away because of the thick tenacious mucous in which they live.

**Life History.**—The eggs of all three species are passed out in the fæces, and development takes place as the result of ingestion of the eggs or larvæ by the larvæ of domestic flies. In the case of microstoma the host is the Stomoxys calcitrans (the stable fly). In the case of the other two, musca domestica (the common house fly). The larvæ escapes from the proboscis of the adult flies on to the skin of the horse, and are swallowed through contact with the mouth. Further development takes place in the stomach of the horse.

The larvæ may escape from the fly when it is resting on small abrasions on the body. In the case of the microstoma, the larvæ may be actually implanted in uninjured skin by the proboscis of stomoxys. In this way larvæ may be associated with the development of summer sores, granulomata, bursatti and conjunctivitis.

**Symptoms.**—As already mentioned, the disease is common in horses in Australia and among Australian horses imported into India. It is common for the three species to be found affecting the same horse at one time.

On account of the severe nature of the lesions caused, which involves perforation and suppurative changes of the lining membrane of the stomach wall on the one hand, and irritation and ulceration consequent upon the presence of myriads of worms on the other, the disease results in marked impairment of condition.

The clinical picture includes the following—History of poor condition over a long period. Debility with progressive-emaciation, in spite of good feeding and a regular appetite. Later anorexia, weakness and incoordination, also anæmia. Colic may be noted where rupture and peritonitis supervene. The action of the bowels appears to be unaffected and the fæces furnish no indication of the presence of disease.

**Diagnosis.**—This must be arrived at largely as the result of the history of the case, and by a process of elimination. As already stated, Australian horses are by far the most frequently affected. The horse should be malleined and examined for the conditions from which differential diagnosis must be made.

In the case of microstoma and muscæ, diagnosis may be confirmed by washing out and examining the stomach contents. The horse should be starved 36 hours, and the stomach flooded with 3 or 4 gallons of water at body temperature. This is then syphoned off, and strained through filter paper, and the sediment examined for the presence of either of the two worms. H. Megastoma, however, cannot be demonstrated in this manner owing to its habit of lying up in the tumours

beneath the gastric mucosa. However, inasmuch as the three species are commonly associated, the demonstration of the presence of one or two raises suspicions of the presence of the third.

Examination of fæces of affected animals may shew the eggs containing the larval forms of the parasites. The eggs of Habronema are oval with a thick shell and are always embryonated.

**Treatment.**—In advanced cases, for obvious reasons, treatment is of little avail. The following treatment has been used extensively with good results.

Starve for 36 hours—an important point—then pump into the stomach the following:—

| | |
|---|---|
| Formalin | ½ oz. |
| Ol. Tereb. | 1 oz. |
| Alœs Barb. | 4 drms. |
| Common Salt | 6 ozs. |
| Ol. Lini | 1 pint. |
| Ol. Chenapodii | 4 drms. |

The above is thoroughly stirred up in a bucket, and warm water added to make the whole up to two gallons.

Allow drinking water *ad lib.* after administration of the above.

Purging sets in after 12 hours, lasts for two days (in some cases there is profuse purging), and there is usually a return to normal on the third day.

The patients should be kept on bran mashes until the third day.

If cases hang fire the treatment is repeated after two or three weeks, and it may if necessary, be repeated as many as six times.

In addition arsenical treatment may be combined with the above, *e.g.*, Ac. Arseniosus in gradually increasing doses 5-30 grains.

It is improbable, however, that H. Megastoma is affected in any way by any method of oral medication.

**Prophylaxis.**—Action must be taken against the intermediate hosts, and breeding of flies restricted as far as possible. Fresh manure should be removed from lines or covered up as soon as possible. Fly breeding places such as dung heaps, garbage, etc. must receive attention. The spraying of dung heaps or the disposal of manure by recognised methods is essential.

### (iii) Strongylidae.

Amongst these the Genera (*a*) Trichonema, (*b*) Triodontophorous, (*c*) Oesophagodontus, and (*d*) Strongylus have to be considered. Among the Trichnonema are included many worms which, when adult, are blood suckers, and which, as larvæ, live part of their life in or under the mucous membrane of the large intestine and cæcum.

The continual plurality of infection has caused the condition associated with the presence of these worms to be known as Sclerostomiasis or Strongylidosis.

**Characteristics of the ova.**—The eggs of Strongylidæ cannot be distinguished from one another. They are oval with thin shells and have a granular central mass which quickly divides in stale fæces resulting in the formation of an embryo.

(a) **Genus Trichonema.**—This includes some 38 species of worms of which the best known is the Cylicostoma tetracanthum, formerly known as the Strongylus Tetracanthus.

*Habitat.*—They inhabit the cæcum and colon.

**Life History.**—The eggs pass out with the fæces, and larvæ are hatched. The larvæ moult twice, and the third stage larvæ which result, retain the cuticle of the second stage as a protective envelope. These third stage larvæ climb up blades of grass and solid objects during the night and on cloudy days. Direct sunlight will drive them down to the soil again.

At this stage the larvæ of the different species have a greater or less degree of resistance to dessication and can remain for considerable periods without food.

The infection takes place by ingestion of the third stage larvæ, which on reaching the cæcum and large intestine (in the case of most species), bore into the intestinal wall and encyst in the mucous membrane. They remain there during the rest of the larval development, escaping from the cysts as nearly mature worms. When the infection is extremely heavy a considerable portion of the surface area of the bowel is rendered functionless. Empty cysts are readily observed on *P. M.*, when the bowel is washed, the surface appearing as though sprinkled with black pepper.

The adult worms lie close to the mucous membrane but do not suck blood. They provoke a brownish mucoid exudate, and take on a red colour as a result of immersion in this exudate.

**Size.**—The female C. Tetracanthus is 10 to 18 mm. long, and the male 8 to 12 mm. As already remarked they may be a reddish colour due to the absorption of pigment of mucous in which they lie in the bowel wall.

**Symptoms.**—An animal of any age heavily infested with cylicostomes becomes debilitated and is incapable of sustained hard work. Such animals suffer from periodic attacks of diarrhœa, at first lasting a few days, then recurring at intervals of a fortnight, becoming more frequent in the later stages, and eventually continuous. The animals become anæmic, and there is excessive wasting of the lumber muscles.

There may be occasional subacute attacks of colic.

Such animals die from debility due to the emaciation resulting from chronic diarrhœa, and probably also to absorption of toxins.

**Diagnosis.**—See later.

**Treatment.**—Oil of Chenopodium and linseed oil, or Ol. tereb and linseed oil. Carbon Tetrachloride is of no value.

Repeat dosage after one month, and again after another month to remove the larvæ which have since developed.

**Prophylaxis.**—Avoid swampy pastures. Young and unfit animals should be placed on an adequate allowance of food. It is doubtful if infection takes place by way of the water supply, as the larvæ cannot swim, and their oxygen requirements are such that they cannot live in water over 6 inches deep.

(b) **Genus Triodontophorus.**—Four species are known to be pathogenic for horses, *viz.*,

T. serratus met with in Egypt, India, East Africa and Mesopotamia.

T. Minor met with in Egpyt, India and West Africa.

T. Intermedius met with in Australia, India, West Africa and Europe.

T. Tenuicollis met with in England.

**Habitat.**—They inhabit the colon and cæcum.

**Life History.**—T. tenuicollis only will be considered. The life history of this worm has been worked out by Dr. Ortlepp. All the details are not yet known but the general outline is as follows:—

The eggs passed in the fæces hatch in about thirty hours. The small larvæ feed and moult twice. At the last moult they remain within their cast skin and are in the infective stage. This takes about four days at 26°C. These infective larvæ can climb up grass, are very resistant to drying, and can survive temperatures from 8°C. below zero to 60° C. above zero. They do not penetrate the skin, and infection is presumably effected by feeding either on grass or on hay. After establishing themselves in the intestine, the larvæ continue their growth, developing a provisional mouth capsule somewhat similar to the adult, and finally, moulting again, become mature. They do not seem to have an extra-intestinal stage such as strongyles have. The other species of this genus probably have a somewhat similar method of development.

**Size.—T. tenuicollis.**—Female 16-19·5 mm. long. Male 13·5 to 19 mm. The other species are about the same size.

**Pathogenic effects.**—T. tenuicollis is responsible in its adult stage for causing serious lesions in the colon of horses. Ulcers are formed in the right dorsal colon. These ulcers may be large and ulcerated, or small and multiple, arranged along the course of the dorsal cœliac artery. There is no doubt as to their pathogenic effect and their blood sucking habits. The worm can invariably be found, frequently in large numbers, in these ulcers and 500 worms have been collected from a single ulcer.

**Symptoms.**—Are unknown but large numbers of adults have been recovered from horses which have died after symptoms of "colic".

**Treatment.**—That advocated in the case of cylicostomes, or strongyles, should be followed: but in view of the fact that the worm gives rise to definite lesions the removal of the worms, even if successful, will not remedy the condition of ulceration to which they have given rise.

(c) **Genus Oesophagodontus.**—Only one species is commonly met with in the horse, *viz.*, Oesophagodontus robustus. This worm has been found in horses and mules in England, Canada and in India.

**Habitat.**—The colon and cæcum, invariably in company with other worms.

**Life History.**—This is not known.

**Size and colour.**—The body is stout, tapering only slightly towards the anterior extremity. During life the worms have a brownish colour and the intestine is deeply pigmented, showing through the semi-transparent body wall. The females are 19 to 22 mm. in length: the males 15-16 mm.

**Symptoms.**—These worms have been observed in large numbers in company with other stronglidæ in mules and horses in India, and there is no doubt that they are at least partly responsible for symptoms of sclerostomiasis exhibited by animals in that country.

**Treatment.**—As for the cylicostomes and strongyles.

(d) **Genus Strongylus.**—There are three species of the Genus Strongylus to be considered, namely:—

Strongylus vulgaris.
Strongylus equinus (armatus or equinum).
Strongylus edentatus.

**Habitat.**—The adult worms are found in the cæcum and colon.

**Life History.**—The life history, as far as phases in the outer world go, is analogous to that of the Cylicostomes. Infection can take place by the mouth and possibly through the unbroken skin. On reaching the intestine, as far as is known, the subsequent history is as follows:—

**S. endentatus.**—This worm then bores through and undergoes part of its development in the peritoneal cavity. It is often found mature in the tunica vaginalis at castration, and at other times free in the abdominal cavity.

**S. Equinus.**—This worm also bores through the intestine and wanders in the peritoneal cavity, and is frequently found encysted in the liver, lungs, and pancreas.

**S. Vulgaris.**—This worm bores through the intestinal wall, enters the mesenteric vessels, and develops, there, the reaction on the part of the host resulting in occulsion of the artery and aneurisms.

Eventually all three species normally return to the intestine and attach themselves to the mucosa, sucking blood. They provoke no brownish exudate as in the case of the cylicostomes and the O. robustus. If washed, they are white in colour, and their intestinal contents brown or black. They are now mature and lay eggs.

**Size.**—S. vulgaris female 24 mm., male 15 mm.
S. equinus female 20-25 mm., male 18-35 mm.
S. edentatus female 33-36 mm. male 23-25 mm.

**Symptoms.**—The most severe symptoms evidenced arise as a result of the complication caused by the migration of S. Vulgaris into the mesenteric arteries.

Sudden attacks of colic, often prolonged, are shewn. Death takes place in an attack of such colic. There is no diarrhœa unless there is a mixed infection with cylicostomes, etc.

Symptoms arising merely from blood sucking habits of the adults will be extremely rare, especially under Army conditions. S. edentatus is a common parasite in the right ventral colon, always present in large numbers and causing great damage to the mucosa. S. equinus is not an infrequent parasite in the same habitat but always occurs in small numbers, S. vulgaris is very frequently present in large numbers in the cæcum.

**Diagnosis.**—See later.

**Treatment.**—Oil of chenopodium may be used, given as already described.

Carbon tetrachloride is a very efficient drug also. The dose is 25 to 50 c.cs. No purge should be given at the time or afterwards.

Phenothiazine has proved to be 100% efficient for the expulsion of strongyloid worms from horses, and Ascaris Megalocephala is satisfactorily expelled.

**Dosage.**—The dose of Phenothiazine is 30 grammes.

**Administration.**—Food is withheld for 12 hours.

The Phenothiazine should be mixed well in a bran mash. The mash is usually eaten but should the horse be reluctant a small quantity of grain (preferably crushed) should be added. After administration food is withheld for 6 hours when the horse can return to normal diet.

Only light exercise should be given during the 48 hours following administration thereafter normal work may be resumed. The dose can be repeated after a month but this will seldom be found necessary.

**Toxicity.**—In the above dose Phenothiazine is practically non-toxic but occasionally slight dullness and inappetence is evidenced. No purging is caused. The urine is usually coloured red for about 3 days after administration due to the presence of the red dye Thional derived from the drug.

In some cases there is a yellow colouration of the conjuctival mucous membranes.

### (iv) Oxyuridae.

The various genera met with in the horse are now believed to be one, and are classified as Oxyurus equi (Curvula and Mastigoïdes). The conspicuous character of the group is the curved anterior portion of the body and the whip like caudal extremity of the female.

**Habitat.**—The adults live in the cæcum and colon.

**Life History.**—The females deposit their eggs on leaving the large intestine and reaching the exterior. The majority stop at the edge of

the anus, being retained by their tails, and the eggs are ejected in a sticky mass on the perineum and under surface of the tail. The female then dies and dries up. The eggs quickly develop on the warm skin, and within 48 hours, generally less they contain an embryo. After 4 to 8 days, the eggs drop off from the skin and with the manure they are further distributed. Water does not appear to be essential for the development of these eggs, but oxygen is necessary. The eggs on being swallowed, hatch in the small intestine and after moulting settle down in the cæcum. Eggs cannot develop without first reaching the air, and this is an essential condition for infection.

**Size of parasite.**—Female 40 to 150 mm. Male 10 mm. As will be noted, the size of the female varies greatly and this has previously led to a double classification of the parasite.

**Symptoms.**—Soiling of the anal region. Itchiness and rubbing of the tail. The worms may produce some degree of debility from irritation caused by wandering gravid females in the posterior colon. The larvæ are active blood suckers and may cause a certain amount of damage in this way.

**Treatment.**—Oil of chenopodium per os. is quite successful. Enemata are not of great value, as all but gravid females are beyond the reach of medicaments so administered. The droppings of affected animals should be disposed of in a proper manner.

### (v) Eustrongylidae.

Species contain Eustrongylus Gigas found in the kidneys of the dog and occasionally in other mammals. The worm has been found up to a metre in length.

### (vi) Trichinellidae.

Species contain Trichinella Spiralis which in the larval form is found sometimes in the muscles of the pig. Man can become infected by ingesting infected flesh.

(b) **Acanthocephala**—

**One species of importance.**—The Echinorynchus Gigas of the pig.

**Habitat.**—The small intestine.

### Annelida.

(a) **Haemopis Sanguisuga.**—The horse leech.

(b) **Hirudo Medicinalis.**—The medicinal leech.

These are "Blood Suckers" and in the process lacerate the skin and mucous membrane to which they attach themselves. They live in water, the young floating near the surface, whilst the adults bury themselves in mud.

They may invade the mouth, nasal passages and pharynx of horses, or attach themselves to horse's legs when the animals wade through infested pools.

**Treatment.**—Removal of those accessible with forceps: with others a strong solution of common salt saturating a sponge on the end of a probe, and brought into contact with the leeches may cause them to loose their hold.

### Worm disease and worm infestation.

95 per cent. to 100 per cent. of Army horses and mules harbour parasitic worms. The mere diagnosis of their presence is, therefore, not of much value in forming an opinion as to whether they are exerting pathogenic effects or not. In other words 'worm disease' must be differentiated from 'worm infestation'.

A diagnosis of 'worm disease' should only be made when all other causes have been ruled out, and when the finding of a pathogenic species is backed up by a typical history and clinical picture. The confirmation of such diagnosis lies in the detection and recognition of the causal parasite, or its ova; and also in an estimation of the probable degree of infestation, *i.e.*, whether it is excessive, above normal, etc.

With this end in view a fæcal examination must be made. Either the parasites themselves or their ova may be searched for, but the latter is by far the more satisfactory proceeding to adopt. Every parasitic worm passes eggs of a constant shape, size and design and knowledge of these is essential for diagnostic purposes.

### Detection of Parasitic Worms.

A Vermifuge test should be applied.

The animal should be prepared by fasting for 36 hours, after which the following may be administered:—

| | |
|---|---|
| Thymol . . . . . . . . | 1½ ozs. |
| Chloroform . . . . . . . . | 3 ,, |
| Ol. tereb . . . . . . . . | 2 ,, |
| Ol. lini . . . . . . . . | 2 pints. |

The fæces should be collected and passed through a sieve, or series of sieves, and the microscopic parasites collected and examined.

### Detection of parasitic ova.

**1st Method.**—The simplest method is to spread a quantity of fæces on a glass slide, dilute it with a small quantity of water or saline, and examine under the 2/3rds objective. Four slides in all should be made and examined, as parasitic ova are not evenly distributed throughout the fæcal mass.

**2nd Method.**—A small quantity of fæces is emulsified in a saturated solution of common salt by shaking vigorously in a test tube. This is then filtered through a small copper wire sieve (100 meshes to the

inch) into a conical flask, which is then filled to the brim with the salt solution. If left to stand for about an hour the eggs will rise to the surface and a wire loop dipped under the surface will remove them to a glass slide. About three loopfuls should be thus taken, a cover slip placed on the drop and the preparation examined as before (Cameron).

**3rd Method.**—Two grammes of the fresh fæces it is desired to examine are weight out and emulsified in about 16 c.c. of clean water. The mixture is strained through a piece of wire gauze having thirty meshes to the linear inch.

The filtrate is divided between two centrifuge tubes and to each an equal volume of sugar solution is added. This solution is prepared by dissolving 1 lb. of sugar in 15 ozs. of water.

The tubes are then centrifuged for two minutes at about 2,000 revolutions per minute in a hand centrifuge.

A piece of plasticine is drawn out into the shape of a pencil and pressed on to a cover glass which has a diameter slightly less than the internal diameter of a centrifuge tube. The cover glass is then lifted and lowered into the tube until its under surface is entirely in contact with the liquid in the tube. It is then lifted out and detached from the plasticine and placed on a slide.

Microscopic examination is then made, using the low power. If desired, a count may be made of the number of eggs in each field. If this is done eight fields should be counted and an average struck.

An idea of the comparative degree of infestation can thus be obtained; for, whereas the normal horse may show an average of 1 to 3 eggs per field by this method, a heavily infected horse may show as many as 9 per field.

The eggs of cylicostomes are not, however, readily distinguishable from those of strongyles and thus the method described above, while of value in giving an idea of the degree of parasitic infection to be dealt with, does not differentiate these conditions from one another. However, in as much as the treatment is the same, this point is immaterial.

**4th Method.**—Another method is to collect a few pellets of dung from a suspected case and place them in a screw top glass jar or other container. The jar if transparent should be wrapped in brown paper and put away in a dark warm place for a few days. After the above interval the jar is opened, the surface of one of the pellets scraped with a scapel and a smear made on a microscope slide previously warmed and moistened with water. Examination will show the presence of larvæ. It is not possible, however, to differentiate these larvæ and the method is merely of use in demonstrating the strongylidæ in general.

# NOTES.

## PIROPLASMOSIS (EQUINE).

**Synonym.**—Biliary Fever.

**Nature.**—Biliary fever is a specific disease affecting horses, mules and donkeys, due to the invasion of the red corpuscles of the blood by a protozoan parasite and characterized by profound alteration of the blood with consequent fever and symptoms of a severe or malignant jaundice, hence the term "Biliary". It is very common in India, and occasions considerable loss. As a rule it attacks only single individuals, but at times it appears to take an epizootic form when there is a large number of susceptible animals.

Formerly the disease was attributed to errors of diet or climatic changes affecting the liver, and it is only within recent years that its true nature and cause have been determined.

**Prevalence and susceptibility.**—The disease occurs in most tropical and subtropical countries. It is more prevalent from April to August, that is immediately preceding and during the monsoon season, than at other times of the year. The reason for this will be explained later on. It, however, occurs at other times of the year, though less commonly.

Imported horses, *i.e.*, English and Australian, are most susceptible, particularly in the very early part of their service or life in the country, and mortality is greatest in them. Arabs shew less susceptibility, and country-breds enjoy a comparative immunity. Horses show the greatest susceptibility, donkeys less, and mules the least.

"Condition" does not appear to have any appreciable influence on susceptibility, the disease being met with alike in well nourished and poor animals and in animals at work as well as those doing no work.

**Protozoology and Infection.**—This parasite until recently was considered to be of one type, but recent observations have proved that biliary fever may be set up by two different types of parasite, *viz.*, (1) Piroplasma (or Babesia) Caballi and (2) Nutallia Equi. Piroplasma Caballi is very similar to Piroplasma Bigeminum (Redwater of cattle). It is usually large and rounded or pear shaped: double pear shaped forms are frequently seen, each element from 3 $\mu$ to 4 $\mu$ in length. Nuttalia Equi is smaller: to commence with, the parasites are similar in shape to the piroplasma parasites, but after a few days other forms are seen which exhibit a peculiar method of division. Within a red corpuscle are seen four daughter parasites arranged radially so as to form a figure like a Maltese cross. Each element is usually considerably smaller than a parasite of the P. Caballi type.

Parasites are not easily found in the blood after the acute symptoms have passed. The disease caused by either type of parasite may appear in the same district and a horse may be infected with both types. Caballi infection is said to occur earlier in the year than the N. Equi infection. In India the Nuttalia Equi infection predominates. The difference in seasonal distribution is due to the fact that the parasites are transmitted by different species of ticks.

The parasites, on gaining access into the system, make their habitat in, or on, the red blood corpuscles, where they carry out their work of destruction. The red blood corpuscles are disintegrated, their number is materially decreased, in some cases even to one-third or less than the normal, and their hæmoglobin is set free in the blood plasma. The blood becomes thin and watery, the excess hæmoglobin results in excess transformation into bile pigment by the liver, and so the train of jaundice symptoms is produced: or the excess hæmoglobin may directly stain the tissues.

The number of corpuscles invaded is comparatively low, rarely exceeding 20 per cent. in severe fatal cases. In bovine and canine piroplasmoses as many as 90 per cent. of the corpuscles may be attacked, and in the later stages of the canine disease nearly all.

The parasite can be constantly found in the early stages of the disease, but its presence is not constant during the whole course of the disease; it is quite possible to diagnose a case clinically as one of typical biliary fever and fail to detect the parasite by microscopical examination. The piroplasm may be found free in the plasma, but such is not common.

Nothing very definite is known as to the manner of multiplication of the parasite, but from analogy in the case of P. canis, a complicated process of division would appear to occur.

It can be detected in unstained preparations with the 1/12 oil immersion lens, but its presence can be more readily determined by simple amiline stains. For practical diagnostic purposes, staining, by a solution of methylene blue (1 in 100) and Eosin (1 in 1000) is the simplest. Mix 5 c.c. of the Esoin solution with 1 c.c. of the Methylene blue solution. Stain for 20 to 30 minutes after fixing in alcohol for 5 to 10 minutes. Or better still, stain with a modification of Romanowski's stain such as Leishman's or Giemsa's.

**Infection.**—The natural mode of infection in all piroplasmoses, so far as is definitely known, is through the medium of ticks. This has been proved so in bovine and canine piroplasmoses and in the equine piroplasmosis of South Africa; but as the latter can be directly communicated by inoculation from animal to animal, there is very good ground for supposing that in India the disease is communicated by other means than by ticks, and that, given a source of supply, any biting insect, such as biting fly or mosquito, can transmit the disease. This has been the experience of our veterinary officers in India during recent years. The disease is more prevalent at times when mosquitoes and biting flies abound, *viz.*, immediately before and during the rainy season: ticks are rare in stabled and well groomed animals, and considerable weight is lent to this supposition of infection by diptera by the fact that malarial fever in men is usually very prevalent at the same time, a common inoculator appearing to be at work.

One attack confers a considerable degree of insusceptibility against a second attack, yet the immunity is not absolute: animals occasionally suffer a second time, but the attack is usually mild. The blood remains virulent, since its inoculation into fresh susceptible animals will at any time produce in them the typical disease. The virus, however, takes an ultravisible form in "immune blood"—unlike the piroplasam of Texas Fever, which remains more or less microscopically visible in the red blood corpuscles of recovered animals.

Incubation after natural tick infection is stated to be 15 or 16 days. By inoculation it is usually about 5 or 6 days.

**Symptoms and diagnosis.**—The cardinal symptoms of the disease are a high temperature and an icteric condition of the mucous membranes. The conjunctival mucous membrane at first assumes a pale yellow colour, which gradually becomes a darker shade until after a few days it is a deep orange or reddish brown colour. Red petechial spots appear, particularly on the membrana nictitans, increasing in size, deepening in colour and finally resulting in large purple blotches. An attack of colic is often a premonitory symptom.

The fever is of an irregular intermittent type. It is highest at the commencement of an attack, 105° or 106° F. There is a marked fall in the first few days, and if the case is mild, the normal is reached by the sixth or tenth day, with or without a secondary rise during that time. In severe cases remissions are fairly frequent and the normal is not reached till about the fourteenth to twenty-first days.

The urine is high coloured, even to deep yellow, from bile pigment. This is however, not a constant sign, and hæmoglobinuria does not appear to have been met with in cases in India.

Constipation is usual at first, the fæces being brownish and coated with mucous. There are also colicky symptoms at times.

The pulse is usually 60 to 80 per minute, weak, irregular, small and wiry.

Urticaria and œdema of the limbs are frequent accompaniments.

When the acute stage of the attack has passed off and the animal is progressing favourably, a well-marked anæmia (from destruction of red blood cells) is seen, the blanched appearance of the mucous membranes persisting for some time.

The course of the disease is very variable, and it exhibits every degree of virulence. It may develop comparatively suddenly, or be ushered in with the usual malaise common to all febrile disease. The attack may be slight or evanescent; it may be short and sharp and lasting only a few days, with rapid convalescence, or it may be protracted and associated with much emaciation. At other times secondary and usually fatal complications such as pneumonia, pleuro pneumonia, extensive pleuritis with effusion ensue, the cause being, in all probability, secondary infection by other micro-organisms.

Provided treatment is adopted in time recoveries are common and loss of condition is not very marked. In untreated cases there is great loss of flesh and frequently severe complications which terminate fatally.

*Post-mortem* appearances are bile staining of the tissues on opening the carcase, thin watery condition of the blood, ecchymoses in serous cavities with excess of serous fluid of a yellow or slightly blood stained character, enlargement, engorgement and bile staining of the liver and considerable enlargement and softening of the spleen. In the complicated cases, the pneumonia is both lobular and lobar, the pleuritis with effusion intense, purulent, and highly fœtid. In other cases there is unmistakable evidence of endocarditis.

## Determination of Red Cell Volume.

A method which has been recommended as an aid to diagnosis is one which is based on the destruction of erythrocytes which takes place in this disease. The method has been practised by Colonel G. F. Steevenson in India who recommends it as simple and efficacious. The following is an extract from his report:—

"The method was used in Macedonia by the Germans during the Great War. It is based on the fact that in Equine Biliary Fever, as in other piroplasmoses, there is hæmolysis and destruction of erythrocytes which causes these to fall below the normal number, to a degree depending on the severity of the disease, *e.g.*, from 7—8 millions per cmm. to even 2—3 millions per cmm.

The method, which is as follows, aims at estimating the degree of corpuscular destruction:—

The apparatus required consists of two similar graduated tubes of about 15 c.c. capacity and fitted with ground glass stoppers. 2 c.c. of a 5 per cent. sodium citrate solution are poured into one tube. The jugular compression pad used for intravenous injection is then applied to the jugular vein of the horse one desires to examine. The needle with the rubber tube attached, as used for intravenous injection (but without funnel), is then inserted into the jugular vein, and the free end of the rubber tube into the graduated glass tube. 10 c.c. of blood is allowed to flow in and then the rubber tube pinched to stop the flow.

There are then 12 c.c. of fluid in the graduated tube, *i.e.*, 2 c.c. citrate solution and 10 c.c. blood. The thumb is placed over the open end of the tube and the tube is inverted two or three times to mix the citrate solution with the blood. The citrate prevents clotting, and the tube is left standing undisturbed for a few hours. All the corpuscular elements then settle at the bottom of the tube, and the height of the column so formed can be read off in c. cs. and 1/10s. of a c.c.

The second graduated tube can, if desired, be used as a control, using the blood from a normal horse.

Observation carried out on 12 normal horses (lame horses) in hospital showed that the height of the corpuscular column varied between 3·2 and 4 c.c., the latter figure being obtained from horses in fat condition which had been resting in hospital for some weeks. About 3·2 to 3·5 c.c. can be considered normal.

In blood taken from a horse suffering from Biliary Fever the height of the corpuscular column is smaller than normal according to the amount of corpuscular destruction that has taken place and varies from just over 1 c.c. in severe cases to 2·5 c.c. or even a little higher.

If blood is examined in the early stages of an attack the supernatant serum is also stained by the hæmoglobin liberated in hæmolysis, and according to the amount of hæmoglobin present, varies from red; a brown colour, to lighter shades. The serum in a tube of normal blood is of course amber coloured.

It appears that hæmolysis is worst in the early stage of the fever and if blood is not taken for examination until the second or third day the serum may be normal in colour through the liberated hæmoglobin having been excreted by the liver and kidneys: the corpuscular column is, of course still smaller than normal. The German workers referred to above also did blood counts with a hæmocytometer and found that if tubes as described were used, that the height of the column multiplied by two, gave approximately the number of corpuscles per cmm. of blood in millions, *e.g.*, if the height of the column, is 2 c.c. then 2 × 2: 4 *i.e.*, the number of corpuscles per cmm. is about 4,000,000 or something like 3,000,000 below normal.

**How to deal with cases.**

1. Remove affected animal into a cool roomy loose box in isolation. The isolation should be as far from unit lines as circumstances and convenience admit. During the acute stages, when piroplasms are frequent in the blood, affected animals are great sources of supply for inoculation of other animals by biting flies or mosquitoes which usually frequent unit lines and stables. These insects are usually very local in their habitat and operations, and isolation is a removal from their sphere of influence.

2. Protection from the sun is very necessary: exposure aggravates the disease; shade of trees and chapper shelter should therefore be provided if isolation is practised in the open.

3. During prevalence of the disease in a unit, any animal off feed should be at once reported, or sent to hospital for examination. Work, while in an unfit state, as in the early stages of the disease, determines a severe case. Mild or recurrent cases occur, and may pass notice. Inspection therefore should be frequent. Inspection of remounts should be particularly frequent.

4. Attention must be paid to the ground surrounding the stables. Pools and other places in the immediate vicinity likely to harbour

insect life should be filled in; inundation should be remedied by proper drainage; manure should not be allowed to accumulate, and manure pits should be at some distance from unit lines. Usually some defect in this direction can be found and easily remedied.

5. The fly and mosquito nuisance should be controlled by every possible means.

6. **Treatment.**—Good nursing is of primary importance. A visit to the patient twice daily should be made, and a temperature chart keep to note the character of the fever.

Easily digested and laxative diet such as bran, linseed, lucerne and sweet doob or green grass should be given. Gruel should be very liberally allowed, and fresh water *ad lib.* Clothe warmly, and sponge body daily with vinegar and water. By every means keep flies and biting insects away. Disinfectants and smelly drugs will effect this considerably. Cheer pine oil or other dressing lightly applied to the face, legs and even on the clothing will be found useful in this respect. Quinine in the early stages is highly recommended, the first dose to be fairly big one. Magnesia Sulphate in three or four ounce doses, until an aperient action is obtained, should be given. Chloride of Ammonia and Belladonna are also recommended; and to assist in elimination of waste products, Nitrate of Potash may occasionally be given. Stimulants are usually necessary, Aromatic Spirits of Ammonia being the most suitable. Complications should be treated according to symptoms presented. Tapping the chest in hydrothorax is not usually attended with success. After convalescence of severe cases, Arsenic and Iron tonics are indicated: gentle exercise in the cool of the day, grazing if possible, and good diet of which oats should form a part, should be allowed.

**Specific Treatment.**—Two drugs have proved successful in the treatment of Biliary Fever in horses, *viz.*, the yellow acid hydrobromide of quinine and tartar emetic. Both drugs are given intravenously and the former is the more efficacious. The quinine treatment, perfected by Brigadier A. J. Williams, D.S.O., F.R.C.V.S., consists of the intravenous administration of one dram of yellow acid hydrobromide of quinine dissolved in one ounce of distilled water into the jugular vein. Greater dilutions may be utilised if considered necessary. Occasionally toxic effects of quinine are experienced, evidenced by faintness or distress; so that it is always advisable to have ready when performing the operation of intravenous injection, a dose of strychnine (grs. ½ to 1) for subcutaneous injection to cambat these symptoms. In severely affected cases it is advisable to give an injection of strychnine prior to the quinine. This treatment was extensively practised in Mesopotamia as a routine measure during the Great War, quinine solution being issued in especially constructed bottles suitable for sterilisation, and for fitting to an intravenous outfit. Treatment was commenced when clinical symptoms were noticed without waiting for confirmation of blood smears. The treatment

met with great success. The salutary effect of this drug treatment is shown in a few hours by a drop in temperature, and the animal commencing to feed.

It is advisable when performing the operation of intravenous injection first to introduce into the jugular vein a solution of sterile normal saline solution, then the drug followed again by sterile normal saline to remove all trace of quinine from the rubber tubing and needle. This is necessary to prevent the introduction of quinine into the subcutaneous or perivascular tissue when the needle is removed, for if quinine percolates into the subcutaneous tissue abscess formation with attendant phlebitis may occur.

**Tartar Emetic.**—This drug is given intravenously into the jugular vein. The dose is from 30 to 60 ccs. of a 1 per cent. solution in sterile distilled water. It is inferior to quinine hydrobromide in that remissions occur more frequently but it is less toxic and preferable in advanced cases. The same precautions as in the quinine inoculation are to be observed to prevent the drug entering the subcutaneous or perivascular tissue. Plasmoquine given by mouth has been used with success. It has the advantage of avoiding the risk of phlebitis but it more expensive than quinine.

Bergthal claims good results in cases due to B. Caballi by injecting 50 c.c. of a 2% solution of trypaflavine into the jugular vein.

**Convalescence.**—The period of convalescence after attacks of Biliary Fever extends from 3 to 4 weeks to as many months. Animals are liable to have relapses if subjected to exhaustive work or inclement weather. Rest and good nourishing food with occasional administration of tonics are indicated.

# NOTES.

## PIROPLASMOSIS (BOVINE).

**Synonyms.**—Redwater. Texas Fever.

**Nature of disease.**—Bovine piroplasmosis is a specific disease affecting cattle, due to the invasion of the red corpuscles of the blood by a protozoal parasite and characterised by profound alteration of the blood with fever and red colouration of the urine, hence the term Redwater.

**Prevalence.**—The disease has a world wide distribution. Babes in Roumania first drew attention to small coccus like bodies in the blood corpuscles in 1888. The parasite was described by Smith and Kilborne in America in 1893 and these authorities demonstrated that the parasite was protozoan in nature and transmitted by ticks.

The disease is common in India and in all tropical and sub-tropical countries.

**Protozoology and Infection.**—The parasite was named the Piroplasma Bigeminum but is more correctly termed Babesia Bigeminum.

The typical form of the parasite of tropical redwater is pear shaped, and they usually occur in pairs, touching at their pointed ends. Each is about 4 $\mu$ in length, and contains cytoplasm which stains blue with any of the ordinary bloodstains, and one or two small chromatin masses which stain bright red. Other forms of parasites occur, *e.g.*, rounded or irregular single parasites cresentic forms, signet ring forms, and single pear shaped forms.

The parasite of European redwater shows considerable difference from that of tropical redwater, although more or less similar shaped forms to tropical redwater are also seen. Immunity set up towards European redwater does not protect against tropical redwater. McFadyean and Stockman named the parasite of European redwater Piroplasma (or Babesia) divergens.

The percentage of corpuscles invaded varies from 1 per cent. to 50 per cent. There is no known method of cultivating piroplasms. The susceptibility of cattle varies according to their age, young cattle being more resistant than adults.

Experimentally the disease can be transmitted by direct inoculation of blood. After recovery an animal may remain a carrier for years. The natural method of infection is exclusively by means of ticks.

**Symptoms and Diagnosis.**—The disease may occur in an acute or chronic form. In a country where the disease is endemic and an animal is a carrier, anything tending to lower the animals resistance such as an attack of another disease may bring on acute symptoms of redwater with fatal results. The symptoms in an acute attack are high fever (106° to 107° F.) staggering gait, a discharge from the eyes, dribbling of saliva, constipation in the early stages, followed by diarrhœa, dysentery, and hæmoglobinuria. The urine varies in colour from light blood tinged to black and there is always albuminuria. The mucous membranes are anæmic or icteric. Pulmonary symptoms may supervene, and death may take place in from 2 to 8 days from the onset of the symptoms.

The chronic form of the disease is more often seen in animals that have been artificially inoculated. In this form fever is intermittent. There is progressive anæmia and emaciation. The mucous membranes may be icteric or ecchymosed. Hæmoglobinuria is not a constant symptom but albumin is always present in the urine. Constipation or diarrhœa may exist, and in milch cows the milk yield is considerably reduced. Death may take place from exhaustion or heart failure.

*Post-mortem* appearances.—The blood is anæmic and watery, the spleen enlarged, the liver is enlarged, jaundiced and shows signs of fatty degeneration. There are petechiæ on the mucous and serous membranes. In an animal that has survived for several weeks the spleen may have regained its normal size.

Diagnosis may be confirmed by a microscopical examination of the blood.

### How to deal with cases.

**Preventive treatment.**—Cattle can be immunised against redwater by the inoculation of a small quantity of blood from a recovered animal. A rise of temperature takes place on the 7th or 8th day and the reaction lasts in some cases for a week. If the reaction threatens to become too severe it can be controlled by the injection of trypanblue. About 100 c.c. of a 1 per cent. solution in normal saline is injected subcutaneously or intravenously. The immunity conferred is not an immunity in the strict sense of the term, since the inoculated animal remains a carrier for the rest of its life and would therefore be a source of danger if introduced into a clean area. The disease is so widespread in certain countries, including India, that it is advisable to inoculate animals before export from Great Britain to these countries.

The eradication of redwater is bound up with the eradication of ticks. The eradication of ticks has been carried out on a large scale in American and South Africa. Eradication entails periodic dipping of all cattle in the given area. The length of the period between dippings must depend on the habits of the particular species of tick, as to whether it is a continuous or interrupted feeder, and how long it spends on the body of its host. Arsenical preparations are the most effective and form the basis of most proprietary dips. Eradication of ticks should also be attempted by burning the grass of places known to be favoured by them when not on their animal host. The freeing of infected pastures from cattle for a prolonged period has also been tried, with the idea that without their natural host the ticks will die out.

Before closing this chapter brief mention must be made of other forms of bovine pirplasmoses, which are of interest as having a wide distribution in most parts of Africa and some other countries, and infections with which are frequently complicated by their co-existence with redwater infection. There are four such diseases, *viz.*, Theileria

Mutants and Theileria Annulata Infections, Anaplasmosis, and East Coast Fever.

### Theileria Mutans.

In South Africa it was found that when imported animals were injected with blood from native cattle, two or three subsequent rises of temperature could be observed, *viz.*:—

(*i*) A rise in from 8 to 10 days due to P. Bigeminum.
(*ii*) A rise in from 26 to 28 days due to Anaplasma Marginale.
(*iii*) A rise in 40 days dues to T. Mutans.

Nearly all cattle in S. Africa are affected with T. Mutans and for the most part it appears to produce little effect, although it is capable of setting up symptoms comparable to milk redwater. T. Mutans is also found in the blood of cattle in India. Exceptionally T. Mutans may cause serious complications and fatal results. These complications are occasionally seen in animals undergoing immunisation against Rinderpest. The parasite is smaller than the B. Bigeminum and very much like the East Coast Fever parasite for which it can easily be mistaken morphologically, the oval, bacillary and comma forms being predominant. As in Redwater the parasite is transmissible by direct inoculation and recovered animals remain carriers.

**Symptoms.**—These consist of suddenly occurring and prolonged fever. The temperature remains very high (105—107) but during the first few days no other symptoms are exhibited. As soon as parasites appear in the blood stream however the temperature begins to waver and fall and rise again, but usually never reaches to height of the early stage. With the appearance of parasites in the blood other symptoms are shown, *viz.*, profuse lachrymation and possibly a watery discharge from the nostrils. Affected animals exhibit pica or depraved appetite to a marked degree. The coat becomes harsh and animals become weaker and lose condition. Superficial lymph glands become enlarged and can be seen distinctly beneath the skin. Affected animals may continue feeding but in spite of this there is progressive loss of condition. Symptoms resembling those of respiratory disease may follow, breathing becomes rapid and apparently painful. This is probably due to anæmia.

Red blood corpuscles may fall to 1/5 of normal. Visible mucous membranes become markedly anæmic and there may be some icterus in the late stages. Diarrhœa may also occur.

The Theileria are not present in the blood in the early stages but appear after the first few days of fever when the percentage of infected red corpuscles may reach as high as 70% with anything from 1 to 10 parasites in a single corpuscle. As the disease progresses the parasites in the blood decrease in number. Koch's blue may sometimes be demonstrated in smears from superficial lymph glands (*e.g*, prepectoral).

**Post Mortem.**—Lesions are not definitely diagnostic and consist mainly in general paleness and anæmia of the tissues, petechiae in the subcutaneous lesions and mucous membrances usually considerable icterus, enlargement of lymphatic glands and spleen, infarcts in the Kidneys and possibly in the lungs. Crater like ulcers with raised edges are almost constant in the abomasum.

**Treatment.**—The most successful drug treatment so far discovered consists of intramuscular injections of Plasmoquine repeated daily for 4 to 5 days if necessary. Intravenous injection of the drug is dangerous. The dose is 32 c.cs. for a full size bull. No other drug treatment should be used with Plasmoquine but every attention should be paid to nursing.

The occurrence and spread of the disease can be controlled to a great extent by short period dipping or spraying with dip. Animals should be kept clean of ticks and every effort made to eliminate ticks from walls and surrounding buildings.

When carrying out short period dipping, caution should be exercised with arsenical dips in order to avoid arsenical poisoning and scalding.

### Theileria Annulata.

Morphological studies on the Indian parasites carried out so far (by Dr. Ray) indicate that Theileria annulata is specifically different from the common innocuous form, T. mutans. The dividing form of the latter were found to occur in the red blood cells of the host, while in the former parasite division took place by means of Schizogony and Schizonts appeared as Koch's blue bodies in the cytoplasm of the monocytes. In cases artificially inoculated with T. annulata by the intravenous route, Koch's bodies appeared in the peripheral circulation within fourteen to twentyone days.

**Histopathology.**—The study of bovine Theileriasis in India and elsewhere has been beset with certain peculiar difficulties. For instance differentiation of individual species of Theileria on the basis of criteria applicable for the purpose among other protozoa has been found to be somewhat unsatisfying. A minute study of the biology of the Theileria parasites in the host tissues has therefore been taken up from the histological and haemocytological aspect. Studies so far carried out show that the predilection seat for the multiplication of the annulata parasite is provided by the lymphoid tissue generally, the disease being primarily an infection of the lymphadenoid tissue. The parasite is present in almost all the internal organs, and infiltration with lymphocyte-like cells in most organs is a constant feature. Widely scattered hæmorrhages in enlarged glands, kidneys and heart muscles are seen, and the hæmorrhages which may assume considerable proportions, appear to be due to parasitised endothelium of blood vessels. Schizonts are more abundant in the liver than elsewhere.

*Diagnosis.*—In view of the above remarks regarding multiplication of the parasite in lymphoid tissue as a predilection seat, in cases where glandular enlargement is present gland puncture smears should be sent for examination in addition to ordinary blood films.

**Technique of Gland puncture Smears.**—Press the fingers between the anterior edge of the scapula and the prescapular gland in a downward and backward direction. With the fingers between the gland and the bone, fix the gland and grasp it in a fold of skin, and puncture with a hypodermic needle with a large bore, 2½ to 3 inches long. Plunge the needle again straight into the gland, then partly withdraw so that the point does not leave the gland however, and plunge again in a slightly different direction. Then withdraw and blow contents of the needle on to a cleaned slide and smear thinly like an ordinary blood smear. The fluid should be milky and not blood stained. With ordinary cleanliness gland puncture does no harm.

### Anaplasmosis.

Anaplasms are minute coccus like bodies 0·1 to 0·6 $\mu$ in diameter and are usually placed towards the periphery of a corpuscle, but are sometimes placed centrally. Hence Theiler distinguished two forms.—Anaplasma Marginale and Anaplasma Centrale. As a rule, from 2 to 15 per cent. of the blood corpuscles are invaded, but occasionally up to 50 per cent. The parasite produces severe blood destruction. It can be transmitted by direct inoculation. The period of incubation is 25 to 28 days. In countries in which the disease is endemic, animals become infected as calves and develop a high degree of immunity. The disease differs from other piroplasmoses in that it is capable of infecting sheep and goats. These animals only suffer from mild fever as the result of infection. Anaplasmosis is a tick borne disease and is rarely seen as a pure infection being usually associated with redwater. The disease is not uncommon in India and has been diagnosed in Diary Farm Animals at Ambala, Dehra Dun, Bannu, Jullundur, Kirkee, Lahore, Lucknow, Mhow, and Rawalpindi.

**Symptoms.**—May be acute and often fatal. There is loss of appetite, rapid wasting, skin becomes dry and yellowish, the mucous membranes icteric, oedematous swellings may appear on dependant parts, there is salivation, constipation followed by diarrhœa the urine dark yellow, never blood coloured. Mortality may be 10 to 20 per cent. Recovered animals remain carriers.

(a) **Treatment.**—A purgative followed by Methyl-Arsenate of Soda (Sodium Cacodylate) of which the dose is 10 to 60 grains intravenously for three days. The drug is dissolved in 4 ozs. of sterile water. Guide to dosage—25-30 grains per 100 pounds body-weight.

(b) **Mercurochrome.**—This drug is also of value in acute attacks. Dose 0·4 gm. in calves and a maximum of 1·3 gms. in fully grown adults, given in a 2 per cent. saline solution intrajugularly. Its use, when advanced anæmic changes are present, is contra-indicated.

## East Coast Fever.

**Synonyms.**—Rhodesian Redwater—Rhodesian Tick Fever—Tropical piroplasmosis.

This is an acute specific tick borne disease caused by Theileria Parva. The disease is widely distributed over Central, East and South Africa, and also exists in Egypt and Transcaucasia. The species of ticks which convey the disease are interrupted feeders. Infection is not carried through the egg of the tick. A tick that feeds on an infected animal as a larva is infective as a nymph, and a nymph that feeds on an infected animal is infective as an adult.

The Theileria Parva is the smallest of the piroplasms and closely resembles T. Mutans. It undergoes development partly in the tick and partly in the ox. In the tick it undergoes sexual multiplication and forms spore-like bodies. These are inoculated into a susceptible animal and take up their residence in lymphatic glands and the spleen. They invade white blood cells and undergo sexual development producing the socalled Koch's bodies. These bodies break up into a number of minute parasites which invade the red corpuscles.

**Koch's bodies.**—Koch's granules or blue bodies are round or oval bodies containing granules in their plasma and are specific for this disease. They are to be found in the spleen and lymphatic glands in the early stages of the disease.

In an acute attack of East Coast Fever up to 75 per cent. of red corpuscles may be invaded by parasites. There appears to be no great destruction of the corpuscles and hæmoglobinuria is not a symptom of the disease. The disease cannot be transmitted by direct inoculation, as the parasites in the blood are non-sexual forms and a recovered animal does not remain a carrier.

The symptoms of the disease are high fever (104° to 108° F.), dullness, cough, weakness, staggering gait, salivation, constipation followed by diarrhœa, swelling of the glands, œdema of the throat, mucous membranes icteric, membrana nictitans ecchymosed, œdema of lungs and pleurisy. The animal may remain on its feed until the disease is well advanced. The disease lasts from 6 to 20 days and the mortality is 95 per cent.

*Post-mortem* appearances are—Yellow colouration of fat, enlargement of glands, especially of neck and throat, blood spots on the surface and lining of heart, serous membranes, gallsac and bladder, œdema of lungs, foam in the air passages which escapes at the nostrils, small erosions or ulcers in the abomasum, blood spots throughout the intestines, infarcts in the liver and kidneys.

The disease has been very successfully combated by short period dipping, *viz.*, every 3 days. By these means the ticks can be completely eradicated.

# NOTES.

# NOTES.

## PIROPLASMOSIS (CANINE).

As Veterinary Officers and Veterinary Assistant Surgeons are frequently called upon for advice with regard to treatment of dogs, a reference to Canine Piroplasmosis, one of the most widespread and fatal diseases of dogs in India, especially of imported dogs, may be made.

**Synonyms.**—Malignant jaundice.—Tick fever.

**Nature of disease.**—A specific disease affecting dogs due to the invasion of the red corpuscles of the blood, by a protozoan parasite and characterised by profound alteration of the blood with fever, jaundice and anæmia.

**Prevalence.**—The disease is widespread in India. It also occurs in Southern Europe, Africa and Asia.

**Protozoology and Infection.**—Two forms of the parasite have been discovered in India, namely, the Piroplasma Canis and the Piroplasma Gibsoni.

The PIROPLASMA CANIS is the larger of the two and it is found in the red blood corpuscles in a pearshaped or ring-shaped form. Multiple invasion of the red blood corpuscles is frequently observed, from eight to twelve parasites often being found in a single red blood corpuscle.

The number of cells invaded depends on the severity of the case, and on the temperature of the dog when blood smears are taken. Occasionally, in acute cases 70 per cent. of the red blood corpuscles may be found to be infected whereas in the chronic from only 3 to 4 per cent. may be infected.

The PIROPLASMA GIBSONI is a much smaller parasite, and often twenty or more of these parasites are found in a single red blood corpuscle. The mortality from this form is very high.

**Susceptibility.**—Puppies and imported doge are much more susceptible to the disease than adult or locally bred dogs.

Different strains of the parasite may vary considerably in virulence, and a dog that has successfully passed through an attack of the disease in one part of India may become severely re-infected if moved to a different part of the country.

The disease is liable to relapses and a dog having successfully thrown off the disease one year may contract it again the following year or earlier, and therefore one attack does not confer any immunity.

**Period of incubation.**—7—14 days.

**Method of Infection.**—Experimentally dogs can be infected by subcutaneous, intravenous or intraperitoneal inoculation of blood from affected or apparently recovered dogs.

Under natural conditions the disease is spread through the bite of a tick, and in India two varieties have been incriminated, *viz.*, Rhipicephalus Sanguineus, and the Hæmaphysalis Vispinosa (Hæmaphysalis Leachhi).

In the case of the first named tick, which is a three host tick, the progeny of the infected female are infective both in the nymphal and adult stage. The infected nymph drops off the infected dog and attaches itself to a blade of grass. If a dog passes, the nymph then crawls on to it, and infects it. After arriving at maturity the tick again leaves the dog and drops on to grass waiting for another dog to pass which it again infects.

**Immunity.**—One attack of the disease does not confer an immunity, and the animal is liable to contract the disease again from a fresh infection, or the parasites lying dormant in some part of the body may spring into activity again should the resistance of the animal be lowered from any cause. Inoculation of the blood of these cases into susceptible dogs will convey the disease.

It has been recorded that the blood serum of a recovered dog contains Anti-bodies, and if such serum is mixed with blood containing the parasites the latter are quickly destroyed.

**Diagnosis.**—For a case to be treated successfully early diagnosis is essential. The only certain method of knowing whether a dog is sickening is by daily temperature taking, and any dog which shows a temperature of over 102° should be treated as suspicious. If any of the symptoms which are described later are noted, blood smears should be taken and examined after appropriate staining (Leishman-Romanowsky or Giemsa).

In dealing with packs of hounds, routine temperature taking should be instituted (*i.e.*, twice weekly) and routine blood examinations of any hounds running a temperature should be carried out.

## Symptoms.

*In acute cases.*—One of the first signs is high fever the temperature being raised to 104° F.—106° F.

The fever is associated with weakness, drowsiness and an occasional symptom evidence is bleeding from the nose.

The mucous membranes of the conjunctiva are at first brick red in colour and the gums have a distinct brick red colouration above the teeth. Later on these mucous membranes become first yellowish in colour, then pale like porcelain, evidencing pronounced anæmia.

The dog refuses all food and often will not even drink water.

If food is given forcibly it is frequently vomited.

Constipation is present at the start, but later on diarrhœa sets in and the fæces are frequently blood stained.

Enlargement of the spleen is not constant, although in chronic cases it is invariably enlarged. The spleen can easily be found on palpation, and if it is enlarged it is hard to the feel.

A most characteristic sign is, that the urine is of a bright yellow colour and stains the floors and wall. Urination is frequent.

The breath has a foul odour and the coat becomes harsh.

The dog rapidly looses condition, and in a few days may be reduced to a skeleton. There is great weakness of the loins, and the dog has a staggering gait. This weakness of the loins is often a permanent feature even when the dog has recovered.

The cornea of the eye may become opaque.

In advanced cases the blood, when shed, is pale and watery. The red blood corpuscles are considerably reduced in numbers, but there is a decided increase in the number of white blood corpuscles. When the disease is advanced, complications such as pneumonia ensue, with considerable infiltration of the lung with a serons fluid, causing death.

*In chronic cases.*—There is progressive anæmia with general lassitude and feebleness. The dog tires very quickly and has an inclination to lie about. On microscopical examination of the blood of these chronic cases only 3—4 per cent. red blood cells are affected. The temperature is not constantly high, sometimes rising to 103°, but with intervals of 4—5 days, during which it remains normal. If the dog is subjected to hard work or exposure, or is not fed on nutritious food, an onset of acute symptoms may supervene with fatal results.

*Post-mortem* appearances.—The mucous membranes of internal organs and intestines are pale or stained with bile pigment. The spleen is sometimes enlarged and the kidneys are congested. The lining membrane of the intestines sometimes shows small hæmorrhagic spots.

When cases terminate fatally, pnuemonia lesions are predominant.

## Treatment.

*General.*—Good nursing, good hygienic surroundings, good bedding, a free supply of fresh air, and no exposure to any great variations in temperature or climate are essential. In the early stages treatment with S. U. P. 36 has given good results.

Good nourishing food, *e.g.*, raw eggs, meat essences, or broth, chopped raw liver, puppy biscuits and rice, etc., are indicated.

Stimulants.—Brandy, port or strong coffee.

If the dog persistently refuses to eat, hand feeding must be resorted to, and food given every three hours.

Free access to fresh water should be allowed.

If penumonia supervenes as a complication, everything depends on good nursing. The bedding should be frequently changed and the area disinfected.

Discharges from the nose should be washed away with a weak solution of potassium permanganate. The gums and teeth should be cleaned with a tooth brush using a reliable tooth paste.

The bowels should be kept open by the occasional administration of epsom salts, 2—4 drms.

If constipation is severe and the mucous membranes are yellow coloured, 1 gr. of calomel should be given with the epsom salts until the evacuations are normal.

*Specific.*—There are several drugs which have been tried, *viz.*, Trypan blue. Novarsenobillon, Sulfarsenol and Tryparsamide, etc.

## P. Canis infection.

### Trypan blue.

This drug is very effective as regards the Piroplasma Canis infection, but is of little value as regards the Piroplasma Gibsoni.

In mild mixed infections it is of great value but in severe advanced cases it is of little use.

The injection can be given subcutaneously or intravenously.

The drug is given subcutaneously or intravenously in 1 per cent. solution in sterile normal saline solution. The solutions should always be made fresh and then filtered through sterile filter paper.

The dose for injection is 1 cc. for every 5 lbs. body weight of the dog. The area selected for inoculation is the groin or neck, and when given subcutaneously the area should be cleaned with ether, and then painted with tincture of iodine. The hypodermic syringe and needle should be carefully sterilised before filling with the Trypan blue solution.

After inoculation the area should be massaged with sterile cotton wool to dissipate the solution. The area of the inoculation should then be fomented every three hours for the ensuing 24 hours with hot normal saline solution. This procedure prevents abscess formation at the seat of inoculation.

## P. Gibsoni infection.

### Novarsenobillon.

This drug is sold in sealed sterile tubes. It should be given intravenously into the external saphena vein, for if given subcutaneously, or any error in the technique of intravenous inoculation occurs it is often attended with serious abscess formation.

The method of procedure is as follows:—Clip the hair round the vein, clean with spirit and then paint with iodine. The needle of the hypodermic syringe is introduced into the vein, the piston of the syringe three quarter filled with Novarsenobillon solution, is withdrawn slightly until blood appears in the syringe, and then gently pressed, to pump the solution slowly into the vein.

The dose of the drug is ·45 gramme, and it should be dissolved in from 5—10 ccs. of sterile distilled water.

The drug usually produces excellent results even in advanced cases. Failure often results when pneumonia and complications have set in, but in chronic cases the effect is extremely good. Even when serious abscess formation has resulted, owing to some of the drug having been introduced into the subcutaneous tissue, good recoveries take place.

These abscesses take a long time to heal as large slough results.

One dose of the drug usually suffices to effect a cure.

### Sulfarsenol.

This drug is as efficacious as Novarsenobillon in acute or chronic cases. Failure results when severe complications have set in.

The great advantage of the drug is that it can be given subcutaneously without risk of abscess formation.

The dose given is dose No. 7—0·45 grammes (45 centigrammes). The powder is sold in small sterile tubes, and should be dissolved in 20 ccs. of sterile distilled water, and inoculated subcutaneously, 10 ccs. on either side of the chest, behind the point of the elbow.

One dose of this drug is usually sufficient to effect a cure.

### Tryparsamide.

This drug has been given an extensive trial by Major Stirling of the Civil Veterinary Department.

The following doses were used :—

Dose—

0·85 gramme for average cocker spaniel.
1·7 gramme for blood hounds.
0·42 gramme for small dogs.

The solution is made up with sterile distilled water.

The drug can be given subcutaneously, the precautions mentioned above for subcutaneous injections being observed.

The drug should be given at intervals of 5 days, and 5 or even 8 injections can be given covering a period of 25 days.

The area should be fomented with normal saline every 3 hours for the 24 hours after the inoculation.

## Prophylaxis.

### Eradication of ticks.

**Area round the kennels.**—Clear the area around the kennels of all grass and burn the grass.

**Treatment of kennels.**—Ticks usually lodge in the upper part of the walls and migrate downwards. A small channel made of tin, in which a solution of Jeye's fluid is placed, should be made about 4 feet from the floor and this trap will deal with the ticks coming down the walls.

The inside of the kennels should be frequently washed down with a reliable disinfectant, and any loose mortar scraped away, as it is in these cracks that the ticks hide. After washing and scraping the whole area, every square inch should be subjected to the flame of a blow lamp, and afterwards the cracks re-mortared.

### Eradication of ticks from the dogs.

When dogs have been out they should be regularly de-ticked, a reliable man being detailed for this work. A systematic search

should be instituted, and with the aid of forceps, the ticks should be extracted and immediately dropped into a receptacle containing paraffin oil and the contents burnt.

The regular weekly dipping of dogs in a solution or a soapy emulsion of Derris will eradicate ticks from dogs. No toxic effects from such dipping are observed. Shampoos and dips prepared with proprietary preparations such as " Soapex ", McDougall's Non-poisonous Sheep Dip Powder, have the same effect.

NOTES.

## RABIES.

**Nature.**—Rabies as it is termed in the lower animals, or hydrophobia as it is known in man, is an acute contagious disease affecting the central nervous system, and characterized by intellectual, emotional and aggressive nervous disorders; otherwise madness. It is mostly seen in canine races (dogs, wolves, foxes, jackals), and is usually communicated by them to man, horses, cattle, sheep, pigs, cats and other animals by biting.

**Bacteriology and infection.**—It is a disease of inoculation, and is conveyed from animal to animal and man chiefly by biting. Animals which use their teeth as weapons of offence, and others whom they can readily attack are the victims.

The specific organism causing the disease has not yet been discovered. It is deemed to be ultramicroscopic. Some bacteriologists consider it as protozoan. It exists especially in the saliva and in the nervous structures of the brain and spinal cord. Saliva is infective two or three days before symptoms of the disease are shown and of course much more so during the progress of the disease.

The infective material, or its toxin, is asserted to proceed to the central nervous system from the wound by way of the peripheral nerves, and not by the blood. Its presence in the saliva is from secretion or excretion by salivary glands.

Though human beings mainly contract the disease by being bitten by dogs and cats, infection may result from handling rabid animals, the poison from the saliva gaining entrance through a scratch or wound. Infection may also result from licking, a rabid animal in the early stages often shewing a disposition to fawn and lick its master.

There is no seasonal occurrence of the disease as was formerly supposed. Outbreaks bear relation to facilities, offered for animals biting each other. Thus in Great Britain and Ireland where the muzzling of dogs was enforced in 1897 there was no rabies from 1902 up to the time of the Great War, when the disease was reintroduced by dogs which were smuggled into the country by aeroplane without undergoing a period of quarantine. A like reduction by muzzling has from time to time been effected in large European cities and districts. For the same reason, in India where there is no proper control of dogs, the disease is very prevalent. Rigid inspection and quarantine have excluded the disease from Australia, Tasmania and New Zealand.

Male dogs form more victims than females (7 to 1), for the reason that they are more aggressive with each other than with females.

The bites of all rabid dogs do not result in the disease. Roughly in animals, experiments have shewn this to be about 50 per cent.

Pasteur places infection in man at 16 to 80 per cent. This wide range in man is greatly due to bites having taken place through the person's clothes, in which case the virus is wiped off the teeth before reaching the skin. Long-haired animals are also protected in a like manner.

Prompt washing and cauterization of the wound protects from infection, and a free flow of blood from the bite may wash out the poison.

So far as is known the virus has not a very long life. Saliva will remain virulent for 11 days if preserved from drying. It is active in water for 20 to 30 days, so that water soiled with saliva may become a means of infection. Saliva dried in a thin layer is soon rendered non-virulent, and brain matter containing the virus loses its virulence in 14 or 15 days when dried in contact with air, and apart from putrefaction. In graves it remains virulent up to 44 days. The virus is very resistant to cold, but steam destroys it within half an hour. Heat at 140°F. renders it inert in one hour, and almost immediately at 158° F.

It is comparatively easily destroyed by caustics and antiseptics. A saturated solution of iodine, strong mineral acids, lime juice, corrosive sublimate and creolin are particularly effective. Lime juice as a convenient, readily obtainable remedy should be noted. Carbolic acid and nitrate of silver are not so good. One per cent. solution of creolin or lime juice kills in 3 minutes; 5 per cent. solution of hydrochloric or salicylic acid, 10 per cent. solution of sulphate of copper and 50 per cent. solution of nitrate of silver in 5 minutes; a 4 per cent. solution of boric acid in 15 minutes; 1 per cent. permanganate of potash in 20 minutes, and a 5 per cent. solution of carbolic acid in 50 minutes.

In view of preventative inoculation in human beings, quarantine and control in the lower animals, a knowledge of periods of incubation of the disease is necessary. These vary with the species, the individual, the seat and character of the bite, and the amount and potency of the bite, and amount and potency of the virus. The nearer the wound to the central nervous system the shorter the incubation. Thus, symptoms are more quickly developed in cases of wounds on the head and face than on the extremities. Multiple and severe lacerated wounds shew a quicker incubation than fewer and less severe wounds.

In the dog, incubation ranges from 15 to 60 days; sometimes it is up to 4 and 6 months. The average is from 3 to 6 weeks. In cats, it varies from 15 to 60 days.

In solipeds, it is usually 20 to 45 days; in cattle 14 to 50 days; in sheep and pigs 14 to 50 days.

In man, it varies between 14 to 64 days.

In rabbits, subdural inoculation has an incubation of 15 days, but by passing through a series of rabbits to obtain a fixed virus for inoculation purposes, the period is reduced to 6 days after fifty passages.

## Symptoms and Diagnosis.

In dogs and other animals, rabies is manifested in two great types, *viz.*, the furious and the **dumb or paralytic.** The former is more frequent in dogs, but each type may merge into the other.

There are **premonitory** symptoms more or less alike in both types, which enable us often to recognise the disease before there is any extreme danger. These are a marked change in the disposition or habits, dullness, melancholy expression, unusual display of affection, licking owner's face and hands, restlessness, tendency to start at the slightest sound, hiding under furniture or in dark corners, morbid appetite,

swallowing all sorts of non-alimentary articles such as straw, cloth, leather straps, etc., tearing sticks, clothes, etc., to pieces, vacant look in the eyes, or mental delusions, the eyes following phantom objects at which the animal will presently snap; the voice may be altered. Dogs should be securely tied up with two chains at this stage, and carefully watched.

In horses and mules simple fever is frequently the only premonitory symptom shown. There is nothing to distinguish this pyrexia from any other simple fever. It may last from 2 to 3 days before any other symptoms of rabies develop. Great care must therefore be exercised when rabies is prevalent or known to exist in a station, in the handling of such cases, particularly as the administration of ammonia carb. balis is a frequent treatment. Frequently colic is a premonitary symptom shown by horses and mules, generally accompanied by a rise in temperature to 101° or over.

**Furious rabies.**—After premonitory symptoms lasting from 12 hours to 2 days, madness is apparent, mental delusions are more marked, a peculiar vacant drawling howl, diagnostic of the disease, is developed, furious symptoms set in, and there is a tendency to bite. Paroxysms of fury may be caused by shaking a stick at the patient or by presenting a dog before his eyes. He will often gnaw his limbs in his fury, or lacerate his gums in biting anything within reach. Salivation is very marked, the saliva hanging like strings from the mouth. There is a tendency to wander at this stage. If an animal gains its liberty, it will travel for miles, biting whatever animals it may meet, flying on them in mute fury. There is no dread of water (hydrophobia) but rather the opposite, as a rabid dog will plunge into water, though it may be unable, from paralysis of pharyngeal muscles, to drink.

Lastly if the animal does not die from the exhaustion of paraxysms in about 3 or 4 days or meet his death from man or animal, he may pass into a paralytic stage and gradually sink.

**Dumb rabies.**—In this, there is omission of the furious stage, the disease merging at once from premonitory symptoms into paralysis. The lower jaw is dropped, saliva drivels, from the mouth, the buccal mucous membrane is a deep violet colour, there is neither desire nor ability to bite. Paralysis extends to the hind limbs then to the fore, and the animal dies in two or three days.

Care must be taken not to confuse the disease with certain other conditions. For instance, a bitch exhausted by lactation may shew delirium with taciturnity and a disposition to bite; a dog struck, or hounded by passers by, may from fear develop a sinister look or a disposition to bite; a bone or other foreign body in the molars may cause an open mouth, dripping of saliva, distress, change of voice, etc.; pentastoma in the nasal chambers, or auricular acariasis will cause distress and a disposition to bite.

The above symptoms are more or less common to all animals. Horses become very vicious, violent, aggressive, mutilating their own bodies,

attacking mangers, stable gear, clothing, etc. They also shew a great desire to micturate, and become sexually excited to a great degree. Cattle shew abdominal pain, stamping, shaking the head, forcing the horns against any object, and tearing up the ground, when sometimes the horns become fractured. There is excessive emaciation and death in about five days. Rabbits, used for the supply of material for inoculation, shew the disease in a paralytic form and exhibit no furious symptoms.

*Post-mortem* appearances do not amount to much in most animals but in dogs the presence of foreign bodies in the stomach, such as straps, cloth, pieces of metal, etc., the result of the morbid appetite, is almost pathognomonic of the disease. The congestion of the fauces, observable during life, may have disappeared after death.

### How to deal with a case; and measures to be adopted in case of animals and persons bitten.

1. Do not jump to conclusions, and destroy an animal on suspicion, or simply because it has bitten other animals or a human being. Tie the animal up and watch carefully for ten days. If rabid, a few days will decide. Segregation in a safe place, and secure tying up, must be insisted on in every case of doubt. Two chains are necessary, one to the collar and the other round the neck.

2. If in 10 days no symptoms of rabies are shewn, the animal may be pronounced free, and no further precautions taken.

3. If symptoms of rabies are clearly developed during isolation, and in every case of the disease met with, in whatever species of animal, destroy as soon as possible after diagnosis. Burn the carcase. Destruction may be carried out by shooting in the larger animals, and in the smaller animals by shooting or poisoning by prussic acid, whichever is most convenient or safe to the person carrying it out. The latter is a most humane, quick, and excellent method, and is to be recommended. Place at least half an ounce of the prussic acid (Scheele's strength) on a pledget of cotton wool wrapped lightly 'round the end of a stick. Present this to the dog through the bars or slightly opened door or his place of confinement as the case may be, or directly to him if securely chained up. In his fury he immediately grabs it with his mouth, and death results. A little more of the acid poured into the mouth completes the process. The sealed up, stoppered ounce vials purchased from chemists are the most reliable.

4. Be most careful to wash the hands thoroughly afterwards with soap and hot water and some disinfectant, such as 1 in 1,000 perchloride of mercury, 10 per cent. solution of carbolic acid, or solution of creolin, cylin, phenyl or whatever is convenient.

5. Thoroughly disinfect loose box or place of isolation. Burn anything contaminated with saliva: pass dog chains, feeding tins and other metal articles through fire. Disinfect walls and flooring with a solution of chlorinated lime, expose to air and sunlight and generally carry out measures stated in "Routine of disinfection" under "General Measures".

6. Enquire and take note of all animals and human beings that have been bitten. Be most careful and thorough in this.

In every case of the disease, bitten animals must be collected. If these are dogs of little value, they should be destroyed under the orders of the Officer Commanding station and the police, the owner being first advised and his permission obtained. More valuable dogs and other animals should be segregated for six months, and periodically observed and inspected. There is no law at present to compel this, or to prevent their removal to another place or district, but usually it will be found that owners realise the danger to themselves and others, and are wishful for advice, and provided that segregation measures are not too irksome and perhaps limited to proper control (*i.e.*, on a chain, or the wearing of a muzzle) when taken out for exercise, will raise no objection. Deliberate opposition should be met with special action by the police.

7. When bitten animals can be got hold of quickly cauterize wounds and dress well with antiseptic, thoroughly washing them first. Pure carbolic acid or sulphate of copper are good caustics. Take plenty of time over the dressing of deep wounds.

8. Report all cases, whether in dogs or other animals, and action taken, to the Officer Commanding station or local Government.

9. Dogs that have been bitten, or are under suspicion of having been bitten, can undergo anti-rabic vaccination, but this should not give a false sense of security to the owner, whose vigilance must not be relaxed during the succeeding period of six months observation.

Persons who have been bitten by or have been in contact with a rabid or suspected rabid animal should report as soon as possible to the local medical authorities. In all cases veterinary opinion should be taken whenever possible and such opinion should accompany the person bitten. If this is not possible or the animal dies before veterinary opinion can be obtained, the brain or portion of the brain preferably the hippocampus major should be placed in a wide mouthed bottle containing a preservative fluid and sent to the District medical laboratory with a history of the case. If human beings have not been involved, the examination of the brain may be carried out by the Military Veterinary Laboratory, Lahore.

The brain should be removed by the method given below :—

**Precautions.**—Use leather gloves over rubber gloves and wear goggles if available as the brain material may be highly infective.

**Method.**—1. Wash the head of the animal with a disinfectant.

2. Fracture the skull on top and sides with a hammer.

3. Reflect the skin, remove the broken bone with forceps.

4. Incise the membranes covering the brain and divide the latter down the centre.

5. Lift out each half of the brain separately severing any nerves or tissues preventing its removal.

**Despatch to Laboratory.**—Send the brain with no preservative if it can arrive at the laboratory within half an hour after removal, otherwise proceed as follows:—

Required for average sized dog's brain, a jar or wide mouthed bottle of two pints capacity. A screw capped jam jar is very suitable.

(*a*) Place a layer of cotton wool on the bottom of the container.

(*b*) Put the whole (in case of small animal) or half the brain into the container.

(*c*) Fill the bottle completely with the necessary preservative fluid.

(*d*) Put a layer of cotton wool in the neck of the container.

(*e*) Cork or otherwise close the container securely, seal with wax and label.

(*f*) Pack in a box with saw dust and enclose an account of the case stating the preservative used.

(*g*) Send by post or passenger train to the Laboratory.

(*h*) Send a separate account of the case by post.

If a biological test (intra-cerebal inoculation of rabbits) is required, send a piece of brain preferably from the hippocampus major in a small bottle preserved in 50% glycerine in normal saline.

Preservative fluids recommended are as follows:—

(*i*) Zenker's solution. A mixture containing—

| | |
|---|---|
| Potasium Bichromate . . . . . | 5 drachms. |
| Perchloride of Mercury . . . . . . | 7 drachms. |
| Glacial Acetic Acid . . . . . . | 21 drachms. |
| Water . . . . . . . . | Ad. 20 ozs. |

(*ii*) Rectified spirits.

Removal of the brain is not necessary if the animal can be certified to be rabid. Symptoms during life are much more reliable for diagnosis than microscopical examination of brain matter; and to determine a diagnosis by the inoculation of rabbits causes delay.

Anti-rabic treatment (Pasteur's) used to consist in the inoculation for 18 to 21 days of millileter doses of spinal cord dried for a varying number of days and emulsified in sterile broth or physiological salt solution. Cords of 14 days' desiccation, therefore weak, were first used, and successive inoculations were from cords of a less number of days drying, the strongest being a cord dried for three days. The inoculation is preventive, not curative, and immunity lasts for at least three years. This treatment has reduced the incidence of rabies from 16 per cent. (at least) to 0·45, or even less amongst Europeans in India.

The system now adopted in India is one introduced by Colonel Sir David Semple, the first Director of the Pasteur Institute of India, Kasauli. The brain of a rabbit dead of fixed virus infection is emulsified in normal salt solution and treated with carbolic acid so as to make a 1 per cent. brain emulsion containing 0·5 per cent. carbolic acid. The virus is killed by this process. The ordinary treatment consists of 14 injections of 5 cc.—one each day.

This method has supplanted the original Pasteurian method, and also that introduced by Prof. Hoyges of Budapest. Vaccines involving a short course of treatment of only 4 days are also employed nowadays.

The following policy with regard to the inoculation and isolation of government animals which have been exposed to infection by rabid or suspected rabid, herbivora or carnivora should be adopted.

(1) Semple's vaccine or 6% Carbolised vaccine will be administered. The carbolized vaccine may be obtained from the Central Research Institute, Kasauli, and when demands are submitted the type of animal (horse, mule, cow, etc.) for which it is required should be specified. Cauterization of the wound alone is not sufficient.

(2) The bitten, animal should be kept in isolation in a Veterinary Hospital for three months.

(3) After the expiry of three months it will be discharged to duty but will be kept under observation for a further period of three months the unit being warned to report any suspicious symptoms at once.

## Prevention of Rabies.

Until general legislative measures for the suppression of rabies are enforced the disease will continue to be prevalent in India; but local measures for the control of dogs in cantonments can be adopted, and will go far to mitigate outbreaks. The following measures are suggested :—

(*a*) Registration of all dogs in cantonments.

(*b*) Taxation—a small fee to be charged.

(*c*) Free issue of a small metal badge on registration, the cost to be defrayed by taxation and the badge to be worn on the dog's collar.

(*d*) All dogs in cantonments to wear a collar provided by the owner.

(*e*) In cantonments, where regimental dogs are exempt from taxation, a regimental register to be maintained, the dogs to wear collars and badges of registration provided at the expense of the owner or regiment.

(*f*) The number of dogs in possession of regiments to be kept at a low minimum.

(*g*) Ownerless or stray dogs to be seized, and if not claimed in 96 hours, to be destroyed. (An ownerless dog to be deemed one without a collar or badge).

(*h*) The appointment of a non-commissioned officer or man for the destruction of ownerless dogs or rabid dogs on the occurrence of the disease.

(*i*) The provision of suitable kennels or cages for the isolation and observation of dogs suspected of rabies, such kennels or cages to be located in a safe place.

(*j*) The issue of instructions regarding care in handling dogs suspected of rabies, *i.e.*, using gloves, provision of proper dog chains, etc.

(*N.B.*—Two chains are recommended, one attached to the collar, and the other round the animal's neck.)

**Synonyms.**—Cattle plague, Bovine pest, Pushims (Hindustani).

**Nature.**—Rinderpest is a contagious fever of a typhoid nature affecting polygastric animals (bovine, ovine, caprine) and characterised by sudden invasion, high fever, extreme infectivity, congestions and erosions of the mucous membranes of the mouth and gastro-intestinal tract, pulmonary emphysema, early and high mortality.

**Prevalence and Susceptibility.**—The steppes of Western Asia, and India may be looked upon as its permanent home, from which it has at different times spread to Europe or other countries with the march of armies, or by extension of commerce. First outbreaks sweep a newly invaded country from end to end, as happened in Africa. It then looses its epizootic character, and assumes that of an enzootic, breaking out in certain centres from which it does not tend to spread.

In India it has probably existed for hundreds of centuries and is now thoroughly enzootic, consequently a certain degree of immunity has been gradually acquired, and mortality is comparatively low. Moreover by a process of weeding out, indigenous cattle have developed a high degree of hereditary resistance. The immunity or resistance is, however, not absolute, and a certain number of susceptible animals keep up the infection. As often happens, the disease dies out in a province, district or village for several years, until susceptible animals increase in number, when an outbreak occurs.

Hill cattle, buffaloes, goats and sheep are more susceptible than plains animals of the same species. The disease in hill cattle is also unusually virulent, the death rate as a rule reaching 90 per cent. and sometimes cent. per cent. It is not nearly so fatal in plains cattle, the death rate in them varying with the virulence of an outbreak, the usual mortality being from 20 to 50 per cent. A high mortality results in Assam and Burma.

Certain breeds shew both a very high susceptibility and mortality. Imported stock from England, Australia and Aden, and all cattle new to the country, are extremely susceptible, and when they contract the disease, 100 per cent. usually die. The Sindh breed is also very susceptible and shows a high mortality.

Cattle are cheifly affected, but the disease may extend to other ruminants. It is more severe in goats than in sheep. Plains sheep rarely succumb. Black buck will contract the disease by ordinary infection.

It has been produced in camels by inoculation of bovine virus and *vice versa*, the course of the disease being mild and terminating in recovery in both instances.

Horses, dogs, rabbits, birds and men, are immune.

Animals recovered from an attack are immune against subsequent infection.

**Bacteriology and Infection.**—The microbial cause of the disease has not yet been discovered. It is ultra-microscopic, and it passes through ordinary Berkefeld filters, but is generally arrested by Chamberland F. Filter.

The blood, secretions, and excretions are extremely virulent, and it is chiefly owing to the diarrhœa, which invariably is present at some stage of the disorder, that infection is spread. The media of infections are numerous. It is conveyed from animal to animal by direct contact, or through the agency of food, litter or water soiled by the infective material. It is also carried from animal to animal and from place to place by flies, by the boots, clothes and hands of persons that have been in contact with affected animals, and birds, dogs, vermin, stray animals, etc. It is improbable that the infective material is carried far by the atmosphere, in fact a deep ditch sometimes limits its spread.

The virus has a short life outside the animal body—at most three days. This should be particularly noted in the view of measures for dealing with an outbreak. It is readily destroyed by drying and putrefaction. For protective inoculation in India, blood from an affected animal cannot be depended on to retain its virulence for more than forty-eight hours, even if kept in an ice chest, unless treated in a laboratory by a special process.

It does not remain active inside the animal body for more than fourteen days, excepting in what are known as chronic bowel cases, but thirty days is a safe limit at which to put the activity in the latter.

The disease smoulders on in a densely stocked location, owing to the occurrence of mild cases in a partially resistant community of animals, which cases may pass unobserved. These, and the chronic cases mentioned above, may start a fresh outbreak if they are brought into contact with fresh or suitable subjects. Virulence, and consequently mortality, is always greatest at the onset of an outbreak the virus either becoming attenuated in action for later cases, or the animals in the presence of the disease developing a partial resistance—a higher rating of opsonic index.

Incubation is from three to eight days. As a rule it is fairly definite at the third or fourth day.

**Symptoms and Diagnosis.**—The first noticeable sign of the disease in a herd is usually the considerable number of animals attacked—a sudden invasion and rapid extension. This rapid infectivity is very characteristic of the disease. In individual cases the earliest symptom is hyperthermia (104° to 108° F.). This rise of temperature is pathognomonic in a herd in which the disease exists, or in animals coming from an infected centre. It usually rises to its height on the 3rd or 4th day of the disease, and goes down below normal before death. In addition to fever there is at first dullness, staring coat, suspension of rumination, mouth hot, mucous membrane of the mouth congested, thirst often great, the bowels generally constipated, the dung covered with mucous, the back arched, and spasmodic twitchings of the muscles of the shoulders, back, and quarters.

Later, on, the gums, buccal membrane and papillae become very red, the tongue furred, the bowels more costive, the dung coated with mucous and blood and passed with straining, the mucous membrane of the rectum and vagina very red and dry, the appetite is entirely lost,

the animal lies down with head turned round to flank, thirst is great, the pulse is quick and irregular, fever is higher, and the twitching of the mucles more marked. As the malady advances, there is profuse discharge of a viscid mucous from the eyes nostril and mouth, the breath is very offensive while epithelial eruptions or concretions, easily detachable and leaving a red surface appear in the inner side of the lips, dental pad and gums. Purging now sets in, the fæces at first being watery and mixed with small hard pellets covered with blood and mucous but afterwards consisting of a fluid matter only with flakes of mucous, blood and aphtha, yellowish brown in colour and most offensive in odour: the abdomen becomes tender. There is great prostration, difficulty in swallowing; the animal is down and has no power to rise; there is a characteristic holding of the breath during expiration, which after a perceptible interval is resumed usually with a moan. Death generally occurs in two to six days.

It should be noted that all the above symptoms are not invariably present, but some of them will always be found. The most characteristic symptoms of the disease are viscid discharge from the eyes, nose and mouth, excoriation and ulceration of the gums and other parts of the inside of the mouth, and dysenteric fæces.

Sometimes eruptions of the skin or epidermic concretions are seen about the dewlap, udder, groin, shoulder and ribs. It is not an invariable symptom, and is generally found in animals attacked in the hot season. It is considered a favourable sign, as when it exists, dysenteric symptoms seldom prevail, and recovery often follows. On this account the disease is considered by some native cattle-owners, to be a kind of smallpox and termed by them " mata ".

**Differential Diagnosis.**—Diseases which may be confounded with rinderpest are :—

(1) Malignant Catarrhal Fever of the Ox.—In this disease there is absence of active contagion, only one or two animals being affected; absence of erosions and epithelial concretions in the mouth; the matrix of the horn is involved; there is great congestion, swelling and discharge from the eyes, and much more serious ocular trouble than in Rinderpest.

(2) Thrush (of the mouth).—In this there is no fever, nor marked abdominal symtoms.

(3) Foot and mouth disease.—The vesicular character of the mouth eruptions, the lesions of the same nature at the feet, no abdominal disorder and the mild nature of the disease sufficiently differentiate it.

(4) Dysentery.—In this disease there is absence of high temperature at first, there is absence of mouth lesions, and the large intestines are affected (*post-mortem*).

(5) Anthrax.—This disease is more precipitate, spreads less widely, lacks mouth symptoms, the bacillus is found in the blood, and there is usually enlargement of the spleen on *post-mortem*.

(6) Hæmorrhagic septicæmia.—In this disease there is absence of mouth lesions, swelling of the throat, death is very rapid. The Pasteurella Boviseptica will be found in the blood or exudates.

(7) Poisoning.—Many points in common. Colic and abdominal pain is marked.

**Post-mortem appearances** are usually significant. Lesions are chiefly concentrated in the fourth stomach (abomasum), small intestines, rectum and oral cavity. In this first organ there is a very deep congestion of the mucous membrane of a dark red or livid colour, with dark voilet and grey stripes and patches of blood extravasation. Ulcerations at the pyloric orifice and on the folds are not uncommon. Sometimes the inflammatory exudate forms a sort of false membrane which can be peeled off.

The small intestines show similar lesions, deep dark red congestions most intense on the summits of the folds, ragged patches of epithelium, erosions, swollen and raised Peyers' patches and the contents of the intestines yellowish grey or reddish, and fœtid.

Lesions in the coecum and colon are less prominent.

The rectum is markedly congested, of a bright red colour, and generally in longitudinal streaks. (The so called " Zebra marking ".)

The spleen is normal, in strong contrast to anthrax.

The liver is generally very friable, and the mucous membrane of the gall bladder is frequently ulcerated and dotted with deposits.

The lesions in the mouth are such as have been described under the heading of symptoms.

The lungs are generally hyperæmic and shew an interlobular emphysema which probably accounts for the arrest in the act of expiration mentioned under the heading of symptoms.

## How to deal with an outbreak.

In India where the disease is enzootic, the guiding principles in the management of an outbreak of Rinderpest should be directed towards—

(I) Separation of the affected from the healthy with the utmost despatch, adopting the group system of (*a*) affected, (*b*) suspicious, (*c*) healthy.

(II) The destruction or treatment of the affected as circumstances or nature of the case warrants.

(III) The protection of all unaffected animals in the herd by the inoculation of anti-rinderpest serum or other methods of inoculation.

1. Remove the affected into isolation as far as the precincts of the farm or lines will admit. Avoid the danger of spreading the disease to other people's animals by traversing public roads. To be of any practical good, isolation must be absolute. If a mile distant so much the better, as temptation of attendants to visit their friends in the lines, thus carrying infection is lessened.

Attendants must undergo strict isolation as well as the animals, and to safeguard against ingress or egress of animals and men the location should, if possible, be fenced with wire rope or brushwood. Food for

animals, and men can be brought by hand to the fence and no further. But see remarks under "Serum alone" inoculation in connection with isolation.

2. Isolate immediate incontacts on either side, in another location, which need not be too far distant, and carefully watch them as suspicious.

3. Inspect and take temperatures of all healthy animals and the incontacts as soon as possible, and repeat daily morning and evening. Any showing a rise is almost certain to be contracting the disease, so remove from healthy group and place in suspicious or affected group according to other symptoms shewn. Include all goats on the premises in the inspections.

4. If possible vacate standings, or lines altogether for a few days, to avoid any chance of infection from them, and to allow of a thorough cleaning and disinfection. [*N.B.*—In this connection the virus will barely live three days outside the animal body, so lines will be soon free.]

5. Thoroughly disinfect standings of affected and incontacts in accordance with instructions laid down in "Routine of disinfection". Pay particular attention to the destruction of litter, fæces, broken fodder, earth of standings, mangers, any article including the clothes, boots, etc., of attendants, likely to have been contaminated by saliva, tears or the stools of the affected case.

Nos. 4 and 5 should certainly be done until inoculation of serum is carried out. If mixing is practised, No. 5 need not be done until animals are again separated—*vide* 'serum alone' inoculation.

6. The veterinary officer called in to the outbreak should at once obtain from wherever it is maintained sufficient anti-rinderpest serum, or inoculate at least all incontacts, if not the whole herd, but this must depend on what prophylactic and protective measures it is decided to adopt. The serum alone method is only useful for tiding over an outbreak. The doses for different breeds are mentioned with the issue of the serum. See remarks under 'Serum alone' method below.

7. **Protective inoculation.**—There are several methods of protective inoculation which may be classed as follows :—

(*a*) Serum alone.

(*b*) Serum-simultaneous method, *i.e.*, virulent blood and serum.

(*c*) Goat virus.

(*d*) Vaccines prepared from internal organs, *e.g.*, spleen, etc.

(a) **Serum alone.**

This method has the advantage that it produces an immediate immunity unless the animal is already infected and the disease in the incubative stage. The dosage is dependent on the weight of the animal and its breed.

To estimate body weight, multiply the square of the girth in inches, by the length, in inches, from the point of the shoulder to the buttock over the ribs and under the hip, and divide by 300. The answer represents the weight in pounds.

The disadvantage of the Serum alone method is that it cannot be depended on to give absolute immunity for a much longer period than ten days; therefore, to protect animals for a month, which is the longest possible duration of activity on the part of the virus within the body (see chronic bowel cases under paragraph Bacteriology and infection), injections should be repeated three times at intervals of ten days. It is safer in the management of outbreaks to consider this shorter period of immunity, especially when such susceptible animals as English, Australian, Aden or Sindh breeds are involved.

To obviate the above-mentioned short immunity, which 'Serum alone' inoculations confer, and the necessity for repeated inoculations, it is advised to mix the healthy animals with the diseased at once after their inoculation. Exposure to infection then results in a mild form of the disease being contracted (often it is only indicated by a slight rise of temperature) and the immunity is consequently active and durable. This system of mixing has now been considered a part of the "Serum alone" method as practised in India. It must, however, be specially noted that until inoculation of the healthy has been carried out, the strictest isolation of the affected must be enforced. It should not be made an excuse for careless dealings with the disease.

Temperatures of all animals under this procedure should be taken daily early at morning time. Any rise in temperature indicates the disease in mild form and active immunity conferred. This (and all recoveries) should be recorded in the animals' descriptive roll for future guidance. An actively immune animal need never be prevented from performing his usual work provided he is kept apart from diseased animals to avoid being a mechanical carrier of infection.

(b) **Serum Simultaneous Method.**—Until the last few years this method was practised as a routine measure amongst Government cattle in India.

The operation consists of two inoculations given subcutaneously and simultaneously—

(1) A small dose of virulent blood, *i.e.*, blood withdrawn from an animal at the height of an attack of Rinderpest during its most febrile period.

(2) A certain fixed dose of immune serum estimated to be sufficient to protect the animal against a clinical attack of Rinderpest.

This has the advantage of producing a lasting immunity in mature animals over two years old.

Calves which should not be inoculated before they are six months old have to be re-inoculated after reaching two years of age as in a proportion of them the protective effect of the inoculation has by then worn off.

The disadvantages of the method are that it entails a great deal of time and labour and is expensive. Large quantities of serum have to be used especially in imported stock, and to enable it to be injected the animal must be placed under restraint.

(c) **Goat Virus.**—Some years ago, goat virus was introduced by Dr. Edwards, Director of the Imperial Veterinary Research Institute, Mukteswar, and was used instead of bull virus in serum simultaneous inoculations. It has the advantages which follow:—

1. The virus is cleansed of the cattle piroplasms to which goats are not susceptible.

2. The virus has a fixed virulence often lower than that obtained from cattle and hence generally safer.

3. Virus can more readily, more economically and without danger be manufactured at the place of inoculation, from the virus obtained from the laboratory. The operator can therefore satisfy himself that he is using potent virus.

Experience has shown however a more important advantage than any of the above, *viz.*, it can be used with safety on cross breed stock without serum being given at the same time and produces reactions which though sometimes severe in young stock are rarely fatal. The solidity and duration of the immunity produced is good and it is not inferior to that produced by s.s. inoculation with "bull virus".

**Method.**—Virulent blood sent from Mukteswar in sealed ampoules is injected into healthy goats for the production of a sufficient quantity of fresh virulent blood for the inoculations. The goats showing a suitable thermal reaction are bled, on the 4th or 5th day after the injection of the virus from the jugular vein into a sterile glass bottle. To prevent clotting of the blood metal strands or glass beads are placed in the bottle to defibrinate the blood by continuous shaking for some minutes after it has been run into the bottle. Clotting can also be prevented by running the blood into one-tenth its volume of a 4 per cent. solution of potassium or sodium citrate.

(*d*) Vaccines made from internal organs.—Tissue virus is usually made from goat spleen, although lymph gland tissue in equally suitable.

**Method.**—Goat spleen tissue vaccine is prepared by slaughtering a goat showing a suitable thermal reaction on the fifth day after the injection of virus, and removing the spleen asceptically. The spleen capsule is incised and small portions of the pulp excised and placed in tubes which are then sealed ready for despatch. Each tube contains approximately ·5 gram and is sufficient for the vaccination of 100 animals. On receipt the pulp should be placed in a sterile mortar and 5 to 10 c.cs. of normal saline solution added, the whole being thoroughly triturated until the tissue is disintegrated. The emulsion is then made up to 100 c.cs. with normal saline solution and filtered through coarse muslin or allowed to stand for a short time in order to remove coarse particles. Dose for cattle is 1 c.c.

This method is not considered as reliable as that outlined in (*c*) above.

The disease is considered to be spread by ingestion of food-stuffs contaminated with excreta (particularly the urine), and nasal discharge of the affected animal. The virus is easily destroyed by natural agencies—the sun, desiccation, etc. This knowledge indicates the extent of the precautions required to prevent the spread of infection.

The inoculated animals should be rigidly isolated, and they should be cared for by attendants who do not come into contact with any uninoculated animal, either directly or indirectly. Strict enforcement of this precaution is absolutely necessary. At the entrance to the building or enclosure where the inoculated animals are kept, a supply of smocks for the attendants should be kept for working hours: if boots are worn these should be changed: if bare-footed a shallow pail of disinfectant should be kept at the entrance for bathing the feet.

### Materials required for inoculation.

1. Special serum for the active immunisation of cattle against Rinderpest by the Serum Simultaneous Method and for the control of, severe reactions after the injection of virus alone.
2. Virulent blood.
3. Hypodermic syringes and accessories.
4. Clinical Thermometers.
5. Microscope with 1/12th oil immersion lens, glass slides, blood stains, etc.

Apparatus for microscope work is essential to examine blood smears of any cattle showing symptoms of Piroplasmosis during the period under observation.

If Piroplasmosis is confirmed it can be readily checked by a subscutaneous or intravenous injection of trypanblue.

It has been proved by experience that a considerable rise in temperature between the 6th and 12th days is practically a sure sign of Piroplasmosis, and that trypanblue should be inoculated in spite of a negative result under the microscope.

### Special serum for the active immunisation of cattle against Rinderpest by the Serum-Simultaneous Method and for the control of severe reactions.

This should be indented for from the Director of the Imperial Veterinary Research Institute, Mukteswar, a short time before the anticipated date of the inoculations. All serum issued from Mukteswar is strictly tested before issue, and is potent at the dosage stipulated on the label on the bottle. The particular brew is also indicated. The serum issued is a special serum from the first bleedings of buffaloes.

As the various breeds of cattle exhibit marked differences in susceptibility towards Rinderpest the dosage for adequate protection must be carefully calculated.

*Buffaloes.*

Dose of virulent blood or tissue vaccine is the same as for cattle.

Severe reactions may be controlled by the use of immune serum.

Disturbances and complications that may appear in inoculated animals:—

1. In cases of severe reaction, purulent discharges from the eyes and nose, anorexia, hyperpyrexia, mouth lesions or diarrhœa may be present. Such reactions should be controlled by the use of suitable quantities of immune serum given subcutaneously.

Hyperpyrexia alone is not a sufficient indication for the use of immune serum.

Ordinarily, an injection of 50 c.cs. of *special* serum per 100 lbs. body weight, subject to a maximum of 300 c.cs., is a sufficient dose to control these reactions.

2. **Piroplasmosis.**—With Goat Virus, the inoculation of Piroplasms with the virulent blood does not occur, but sometimes the inoculation reduces the natural resistance of the animal and dormant piroplasms present in some remote part of the body are stimulated to increased activity. The presence of these piroplasms will affect the animal considerably, and cause a sharp rise of temperature between the sixth and tenth day after inoculation, together with malaise and perhaps hæmoglobinurea.

If this complication is experienced, treatment should be adopted forthwith by injecting 100 c.cs. of a 1 per cent. solution of trypanblue either subcutaneously or intravenously. If a fall of temperature does not occur in six hours and the constitutional symptoms become more serious the injection should be repeated, this time intravenously.

3. **Theileriasis.**—Similarly to Piroplasmosis, the inoculation sometimes brings out a latent infection of Theileriasis.

**Treatment.**—An intramuscular injection of Plasmoquine should be given as soon as the diagnosis is certain; this may be repeated daily for 4 to 5 days if necessary. Laxatives and good nursing are also indicated.

**Coccidiosis.**—The sign of this complication occurs usually a few days after the normal Rinderpest reaction has passed, when the animal will begin to strain, show colicky symptoms and evacuate masses of pure blood clots with the fæces. The fæces then assume a watery consistency.

When this condition is diagnosed, gruels should be administered and mild internal disinfectants, intestinal astringents and general stimulants should be given.

**Treatment of Rinderpest.**—This is prohibited in many countries, but in India where the disease is so general, and where a good percentage of recoveries results, it should be carried out. Usually, too, at the commencement of the outbreak so many animals are found affected that wholesale destruction is out of the question. Therefore, treat all cases, but do so in the strictest isolation giving first an appropriate dose of serum. Nursing and proper diet are essential to successful treatment. Keep the body warm with sufficient clothing, and place animals in a place sheltered from rain or from exposure to sun. Diet should consist of rice gruel well boiled and of good consistency. Water may be given in the first stage as long as the bowels remain costive, but when purging

has set in only luke-warm water in small quantities, and at short intervals, should be given. Gruel is preferable at this stage, and the animal's thirst, which is often intense, should as far as possible be satisfied with it instead of with water. Epsom salts in 2 ounce doses twice daily, or very small doses of calomel (20 to 30 grains), as an internal antiseptic repeated twice daily in early stages until diarrhœa sets in, will be found beneficial. The mouth should be frequently gargled with Boracic acid lotion and care should be exercised with regard to the mouth becoming fly-blown.

When purging is arrested, and the animal shews signs of recovery, a little green food or lucerne may be given. Common salt in small quantities in the gruel during the disease, and in the food or hung up to lick, after recovery, is beneficial.

The duration of the disease is from 2 to 12 or 16 days generally three to nine days.

Carcases should be incinerated or deeply buried, the hides being previously slashed.

During treatment the continued disinfection of the location where animals are isolated must be practised, a special bath for the disinfection of attendants, their clothing, boots, hands, etc., being maintained. This cannot be too often or too strictly insisted on as one of the most important factors in the management of the outbreak.

9. **When to declare the outbreak at an end.**—Strictly speaking a unit cannot be considered free from the disease until one month (30 days) after the occurrence of the last case—the very outside limit of infection; but if affected cases are strictly isolated, and the remainder subjected to protective inoculation by the "serum alone" method, by which immunity is immediately conferred, there is no reason why the latter should not return to duty after allowing for a reasonable period of incubation, *plus* a few days' extension which the serum may cause. After 10 days from the last case, therefore, the healthy animals of a unit may return to duty, performing their duty under "working isolation". Any undue restrictions, when proper protective measures have been adopted, mean a monetary loss to Government in the hiring of animals.

When mixing is practised the majority of animals will have either passed through a mild form of the disease or shewn themselves immune in fourteen days. These can then be drafted out, thoroughly washed and disinfected, and then be sent to duty in working isolation.

In military dairy farms the same thirty days' rule applies. No hardship is entailed, the animals can be entirely stall fed for that period, and the milk of unaffected animals (*i.e.*, no increase of internal temperature) is quite fit for use.

### Prevention of outbreaks in military service.

This is very important, and more attention should be given to it than appears to be done—

(1) Isolate all newly purchased cattle and buffaloes from fairs, villages, etc., for at least 14 days. Arrange for a place well away from unit lines.

(2) When the disease is known to be prevalent in the district restrict the movement of private cattle, buffaloes and goats through or in a Cantonment. Consult with the Officer Commanding station and the Cantonment Magistrate regarding this and work in conjunction with civil authorities. Much good can be done by co-operation with the latter (Civil Veterinary Department).

(3) When unprotected transport bullocks are under orders to march through a district known to be affected with Rinderpest, inoculate all with serum previous to march.

(4) A more practicable and better system is to confer durable immunity by inoculation at the commencement of their career in military service.

### Rinderpest on active service.

Prior to proceeding on active service all bullocks should be inoculated to give them an immunity to the disease.

# NOTES.

**Nature.**—Strangles is a febrile catarrhal inflammation of the upper air passages, with formation of abscesses in the adjacent lymphatic glands, in other lymphatic glands, and in the skin.

**Susceptibility.**—It is a disease of horses and mules; donkeys are more rarely affected. Other animals are naturally immune.

Youth predisposes to the disease; it is most commonly seen between two and five years of age, but it frequently attacks foals even a few weeks after birth. Old animals, if they have not had the disease previously may contract it. One attack usually confers immunity, though such is by no means constant.

**Bacteriology and infection.**—Until recently strangles has been considered to be due immediately to the streptococcus equi of schutz which abounds in the pus of abscesses, and in the catarrhal discharge from the nose. The organism is also found in the alimentary canal and in the blood to a limited extent. It has been claimed, that in common with influenza, catarrh, sore throat, and contagious pneumonia, the disease is started by the pasteurella cocco-bacillus, only discoverable in the early stages; this organism paves the way for the streptococcus, the former reducing the resistance, the latter causing the disease. It is also possible that as in canine distemper the primary factor in the causation of strangles may be an ultra-visible virus.

The streptococcus is easily found in the pus by the microscope, stains readily with aniline colours so that it stands out clear among the pus cells. It appears as chains of cocci, two to four or more in a chain, the chains being straight or sinuous. It has a saprophytic existence: infected soil can not only harbour it, but can multiply it. It seems to hang to certain stables, infection being kept up by the occurrence of cases at intervals. Healthy horses from such stables can carry the infection for some time, and can transmit it to a less resistant neighbour. It will remain virulent in the bowels of a recovered case for months; thus manure is a source of infection and soiled fodder and litter are dangerous. It is impossible to say how long it can live as a saprophyte, but it is certain that it requires a host to continue its existence. It is easily killed by disinfectants when accessible; therefore disinfection measures keep it in check. Modern research would tend to support the view that the so-called streptococcus of strangles is not the primary casual agent but that the casual agent is probably a filterable virus. What lends support to this view is the fact that all attempts to produce a protective serum or a vaccine by means of the streptococcus have failed.

In addition to youth as a predisposing cause, chill, change of surroundings or locality, overcrowding, fatigue, dentition, a catarrh of air passages all favour an attack.

Infection is usually through the mucous membrane of the nasal chambers by inhalation. Next to inhalation the most prominent channel is through castration or other wounds. The infection through castration wounds, with subsequent peritonitis, is much more common in mules than horses.

Incubation is from 4 to 8 days.

**Symptoms, types of the disease, and diagnosis.**—The usual or **regular** form of the disease presents a certain amount of fever, a discharge from both nostrils at first watery, afterwards cloudy, and lastly thick, opaque and mucopurulent; a diffuse swelling of the submaxillary glands and surrounding tissue, quickly developing into an abscess, usually large, with a tendency to burst, discharging pus of a white creamy nature.

During an outbreak, cases shewing catarrhal symptoms only, without the submaxillary abscess, and others shewing the submaxillary abscess without catarrhal symptoms are quite common.

**Irregular** forms of the disease occur. These include:—

(*a*) Pharyngeal and laryngeal strangles, with swelling of the throat, laryngitis, difficulty in swallowing, abscesses originating in the guttural lymphatic glands sometimes bursting internally but oftener giving trouble in external operation owing to the proximity of the parotid salivary gland and the large arteries supplying the head. This condition may be secondary to a regular case of the disease.

(*b*) Abdominal form, with abscesses in the small lymph glands of the intestines or mesentery, infection being either carried by the blood from an original centre, or succeeding castration. The latter is not uncommon in India.

(*c*) Pulmonary complications, pleuritis, pneumonia and strangles abscess in the mediastinum.

(*d*) Secondary abscesses in the brain.

(*e*) Abscesses elsewhere.

(*f*) Small pustules and abscesses on the skin of face running towards the submaxillary gland: or on the legs and the hollow of the heels—a cutaneous strangles.

**Diagnosis.**—The disease may be mistaken for glanders, particularly if there is excoriation of the mucous membrane of the nostrils as frequently happens. The excoriation is not ulceration as met with in glanders, and besides the abscess under the jaw bursts in strangles, whereas the swelling at the same site in glanders is hard and does not burst.

Cutaneous strangles may be confused with Epizootic lymphangitis or Farcy, but examination of the pus by microscope shews the streptococci of strangles in great numbers.

In both cases there is no reaction to Mallein.

## How to deal with an outbreak.

1. Remove case from lines and isolate completely.
2. Burn contaminated litter, broken forage, dung of affected animals. Particularly destroy sponges or rubbers used for cleaning nostrils.
3. Thoroughly disinfect standing, manger or any feeding or watering utensil used by affected animal.
4. Include standings of animals on each side in the process of disinfection.

5. If several cases have occurred, vacate the whole stable, clean and disinfect it thoroughly. Include the attendants in the disinfection.

6. **Treatment**—mainly hygienic and antiseptic—antiseptic inhalations, or insufflation, operation on abscess, hot water applications in laryngitis cases. Carefully burn the pus from abscess, also tow, cotton wool and other dressings that have been used. Use cotton wool or tow to clean nostrils and destroy after. Be most careful to sterilize instruments used. Destroy all dung, soiled bedding, and rejected food of cases under treatment. Treatment with Sulphanilamide is under trial.

7. Do not perform castrations during an outbreak nor on an animal that is suffering or has recently suffered from the disease.

8. Be in no hurry to return a recovered case to troop lines, especially amongst young horses, remembering that it is a carrier for some time.

9. Attend to general hygiene of lines, drains, water troughs, etc., as a prevention of spread.

**Prevention of introduction.**—All animals, and particularly young animals, should undergo one month's complete segregation before joining a unit. Any animal with discharge from the nose should be kept by itself, and have its own watering, feeding and grooming kit.

The existence of Government piggeries, and the possibility of the outbreak of contagious disease in them, necessitates the inclusion of a short description of swine disorders in this handbook.

For practical purposes the various contagious diseases of swine are grouped under the one generic term of "Swine Fever", and include the following well-known forms:—

(1) Swine Erysipelas (Great Britain) or Rouget (France).

(2) Swine Fever (Great Britain), Hog Cholera (U. S. A.) or Schweine pest (Germany and the continent of Europe).

(3) Swine Plague (U. S. A.) or Schweineseuche (Germany and the continent of Europe).

**Nature.**—The above are all fevers of an eruptive type, highly contagious, and attended with great mortality. Though certain manifestations are common to all, particular symptoms and the presence in each of particular microbes resulted in their classification as separate diseases. Recent investigations, however, appear to show that Swine Fever and Swine Plague, at least, are one disease, caused primarily by a micro-organism which is ultravisible to the highest known power of the microscope, and that the hitherto reputed causal micro-organisms only play a secondary role in causing the particular symptoms which have been held to differentiate the diseases. At some future time the disease known as Swine Erysipelas may also be brought into line with this view.

**Symptoms and diagnosis.**

(1) **Symptoms common to all.**—Loss of appetite; disposition to separate from its companions; animal buries itself under litter; squeals if moved; staggers about; temperature elevated to 105° F. and upwards [the normal temperature of pigs is high, varying from 100° F. in a cold draughty pen to 104° F. in a warm dry location with plenty of exercise (Law)]; congested mucous membranes; eyes watering, with a mucopurulent exudate gumming eyelids together; often great thirst; vomiting; a red rash on the skin, especially on the abdomen, inner side of legs, breast, and ears, the red colour having a tendency to become blue.

(2) **Particular symptoms and diagnosis:—**

(a) **Swine Erysipelas.**—Rapid onset, incubation one to three days, mucosæ deeply congested even to violet colour; temperature exceedingly high, 107° to 109° F.; more extensive petechiæ on skin; attacks mature swine generally; death common within 48 hours; sometimes rapid recovery, though mortality usually averages 80 per cent.; spleen found enlarged and soft on *post mortem* examination.

(b) **Swine Fever or Hog Cholera.**—Incubation six to fourteen days usually; **enteric symptoms,** constipation with glazed dung, followed by profuse, watery, fœtid, bloody, black or yellow diarrhœa; vomiting common; enlargement of lymphatic glands, best felt in inguinal glands;

death after one or two weeks or more; absence of cough as distinctive from swine plague. *Post-mortem* examination shows ulcerations on mucous membranes of intestines, especially in cœcum and around the ilio-cœcal valve, the ulcerations being diptheritic, laminated and from proliferation of the tissues underneath, assuming a button shape. The spleen is little affected as in swine erysipelas.

(c) **Swine plague.**—Incubation short, sometimes one day; very high temperature, up to 109° F.; greater reddening of visible mucous membranes; comparative absence of abdominal tenderness and bowel symptoms; constancy of cough, dyspnœa, and symptoms of broncho-pneumonia; rapid emaciation.

On *post-mortem* examination lobular and lobar pneumonia affecting chiefly the anterior lobes is seen, with necrotic foci and points of pus in the air sacs and smaller bronchi. The spleen is little altered, and there is a comparative absence of ulcerations in the bowels, especially the button-shaped sloughs of hog cholera.

### Bacteriology and infection.

The ultravisible virus, to which is attributed the primary cause of Swine Fever, is so small that it passes through the closest grained porcelain filters; and it is not cultivable outside the animal's body. It appears to be non-pathogenic to other animals than swine.

Experiments show that it is still active after keeping for ten weeks at ordinary room temperature. A temperature of 172° F. destroyed it in one hour, and putrefaction in one week.

Under the diminished resistance of the body caused by the ultravisible virus, the so-called "Hog cholera bacillus" (Bacillus choleræ suis), formerly the reputed cause of Hog cholera, is responsible for the bowel symptoms of that disease. It is an actively motile bacillus, with rounded ends, usually found in pairs, and staining readily with aniline dyes. It is probably a normal inhabitant of the pig's intestines.

Experiments in America have shown that it has considerable vitality outside the body. It can be kept alive in clear river water for from two to four months, and it remains active in soil for the same period.

Under like circumstances the pulmonary manifestations of Swine Fever are due to the so-called Bacillus of Swine plague (B. Pestis suis), the formerly reputed actual cause of Swine plague. It is a short bacterium, non-motile, staining distinctly polar, and showing much less vitality than the hog cholera bacillus. It is killed by drying in three days, is instantly destroyed by disinfectants, and remains active for 4 to 6 days in soil or for 10 to 15 days in water. It closely resembles a bacillus found on the nasal mucous membranes of healthy pigs.

The germ at present reputed to cause Swine Erysipelas is a short rod, non-motile, found only in small numbers in the blood (in the leucocytes), but abundantly in the lymph glands and spleen, and enormously in the urine and fæces, particularly the former. It is easily destroyed by drying, and by disinfectants.

**Infection.**—Infection probably always results through the digestive canal by the ingestion of food or earth soiled by the excreta of affected animals. A strange pig brought to the piggery may commence the disease, or it may be introduced through the medium of the diseased refuse products of slaughter houses which serve as food for the animals. Once introduced the habits of the animal make the spread of the disease in the piggery readily understandable.

Infection is also kept up in a herd by cases which never develop any decided symptoms. These chronic cases are equally contagious, and may be recognised by an unthrifty look, a capricious appetite and an irregular action of the bowels.

Certain conditions favour infection. These may be summed up in:—

(*a*) In-breeding and importation of highly bred stock, which are always more susceptible.

(*b*) Debility from injudicious feeding.

(*c*) Filthy surroundings, with dark pens and insanitary bedding.

(*d*) Over-crowding, with greater facility for eating each other's droppings.

A prevalent idea is that anything is good enough for pig's food. Kitchen swill, consisting of soiled and spoiled food, and probably containing carbonates and bicarbonates of soda, which in themselves are deadly poisons to pigs, the waste products of creameries reeking with toxins and ptomaines, salt in excess, too much cotton seed, all induce gastric and intestinal disorders, and indeed often produce symptoms closely resembling swine fever itself.

With regard to housing it should be noted that the pig, as partly explained by its high normal temperature, requires twice the breathing space for every 100 lbs. of its weight, than that required by either horse or ox.

## How to deal with an outbreak.

1. Report as early as possible. Instant action is necessary.
2. Destroy all affect and incontacts, the incontacts to be considered those which have occupied the same pen or location as the diseased.
3. Remove the animals of unaffected pens or locations as far away from affected pens as the precincts of the premises will admit of, and isolate them as completely as possible, particularly remembering the danger of attendants conveying the disease. Spread the animals out as much as practicable.
4. If infection is general, it is best to make arrangements for the destruction of the whole piggery, and dispose of the animals for food while they are healthy. The death rate is 80 to 90 per cent. in acute cases, and 50 to 60 per cent. in less acute cases. The hotter the weather, the higher the death-rate.
5. Burn all affected carcases.
6. Burn all litter and manure of affected pens.
7. Collect all excrements on the premises daily, and burn.

8. As soon as possible thoroughly disinfect buildings, yards, fences, drains, utensils, and any article likely to have been contaminated. Include all attendants in the process of disinfection. Pay particular attention to contaminated ground. For procedure see "Routine of Disinfection" under "General Measures".

9. Prohibit all movement of animals within the premises, excepting those carried out as indicated in paragraph 3.

10. Prohibit visitors to the premises, and exclude domestic animals and birds as much as possible. Keep premises as free from flies as season permits.

11. Treatment is deprecated.

12. Keep a careful look out for any unthrifty looking animals, or any showing indefinite enteric symptoms, or symptoms of pneumonia.

13. Boil all food and see that it is fresh and wholesome and well balanced as a ration.

14. Attend to the hygiene of the healthy animals, and with dry warm beds, plenty of air and light, maintain them in good health and vigour.

15. Discard the long feeding trough.

16. Pens to hold small numbers, or a wide enclosure, so that they can escape from their own filth, are advisable. Pollution of streams should be avoided.

17. Infected pens or premises should not be re-occupied for six months.

# NOTES.

NOTES.

# TETANUS.

**Synonym.**—Lockjaw.

**Nature.**—Tetanus is a disease of animals and man characterized by tonic spasms of the voluntary muscles in a given region of the body or more generally. It is not a contagious disease in the ordinary acceptation of the term, spreading from animal to animal like glanders or rinderpest; but is usually met with in isolated cases, is more prevalent in tropical countries than in temperate or cold ones, and in some localities more than others.

**Bacteriology and infection.**—It is due to the Bacillus Tetani, and infection is through the medium of wounds. The bacillus is a feebly motile, rod-shaped organisms 4 to 5 microns in length and 0·4 micron in thickness with rounded ends, one end being enlarged by the formation of a spore, which gives the bacillus the characteristic shape of a drumstick. It grows only in absence of oxygen (anærobic).

The bacilli and their spores are saprophytes, widely distributed in nature, and found in soil, dust, water, manure, drains, cracks in floors, and in the alimentary canal of healthy animals. Only when implanted in a wound can they give rise to tetanus. They flourish best in deep, punctured, lacerated wounds. They remain strictly localised in the infected wound, manufacturing a poison (tetanin or tetano-toxin), which gives rise to the well known symptoms of tetanus. The toxin has a great affinity for nervous matter, and operates through the nervous system, travelling along the peripheral nerve trunks to the central nervous system. In large doses or severe infection, the toxin may, in part, be carried by the blood stream but even in that case it is taken by the nerve endings of the blood vessels and carried along the nerves to the brain and spinal cord.

The spores have a wonderful vitality: they survive desiccation for years, and can live 2½ months in putrifying material; they resist a temperature of 176° F. in water for one hour, but are killed by boiling (212° F.) in four minutes. They are very resistant to chemical agents. The bacilli are killed by a temperature of 140° F. to 149° F.

The organism and spore stain readily. It is satisfactorily demonstrated by Ziehl's method. Float a cover glass smear in carbol-fuchsin (1 part of a 10 per cent. solution of fuchsin and 10 parts of a 5 per cent. solution of carbolic acid), heat for three to five minutes till steam rises, decolorize in 25 per cent. solution of nitric or sulphuric acid, afterwards in 60 per cent. alcohol, wash and counterstain with a solution of methylene blue. The bacillus is stained blue and the spore red.

The blood and tissues are not infective, but pus from an infected wound is.

**Methods of Infection.**—Occurs as a rule through the contamination of wounds of the skin or mucous membrane with dirt containing bacilli. The most common medium is earth, and the most favourable wounds are punctured ones, or injuries associated with considerable destruction of tissue. Wounds which come in contact with the earth, or dung, are

most liable to become infected, such as wounds of the feet, surgical wounds following operations such as castration and docking.

Idiopathic cases are those in which no visible wound can be found. Such cases may result by infection of the buccal or intestinal mucous membrane. The bacillus is essentially a tissue parasite and remains at the seat of entry, multiplying and elaborating the toxin. The presence of suppurative organisms favours the growth of bacilli.

**Period of Incubation.**—In the horse from 3 to 15 days. In the ox and sheep may be as short as 2 days.

**Symptoms and Diagnosis.**—During the first day or two the animal is disinclined to move and has a sluggish action when forced to do so. The legs appear stiff and do not flex normally when the animal moves. The patient masticates slowly and has difficulty in swallowing. There is a peculiar projection of the head, and a rigid stance. Later, spasms occur, which may start in the head or hind quarters and spread either backwards or forwards. In some cases the spasms are confined to certain definite areas. The pupils are dilated and the eyes drawn into the orbital cavities owing to spasm of the eye muscles. The membrana nictitans projects across the eye to a greater or less extent, which is particularly noticeable when the head is raised or the animal given a tap under the chin. The spasm of the pharangeal muscles prevents the swallowing of saliva which hangs from the mouth in long threads. The abdomen is contracted, the tail rigid and sticks straight out or is drawn to one side. Some muscles of the body are sharply contoured and sometimes show fibrillary tumours. On palpation the muscles are hard and board like. Reflex irritation is greatly increased, the appearance of a light, a touch, or the slightest noise bringing on a spasm, the spasm usually being accompanied by profuse sweating. Respirations are superficial and rapid and there is an incomplete change in the gases resulting in cyanosis. The internal temperature is only increased by 1° or 2° but shortly before death when spasms are almost continuous the temperature may become elevated to 105° or 107°.

The disease may be confounded with strychnine poisoning, rabies, rheumatism of the neck, and meningitis. In strychnine poisoning, the spasms are more suddenly developed, not confined to a particular group of muscles, but general, more intense and with intervals of complete relaxation or clonic spasms. In rabies, there may be history of a bite; there are always hallucinations and a mischievous disposition; the spasms are clonic, there being intervals of complete relaxation; and in advanced rabies there is paralysis.

In rheumatism of the neck there is no trismus and no marked hyperæsthesia. The same may be said of meningitis.

## How to deal with a case or cases.

Excepting in very severe, hopeless cases, treatment should be attempted. If incubation is short, and symptoms set in violently, death

usually occurs within a week. If incubation is over one, two or three weeks and symptoms are equivocal or slight, with little projection of membrana nictitants, or the jaws not entirely closed some days after onset, or there is trismus only, treatment is favourable. The mortality in horses is about 75 to 85 per cent. In cattle the mortality is about 70 per cent. : the disease in them is usually slow, and improvement may not begin until the third week. In sheep, goats and dogs (the latter very rarely contracting it) the disease is habitually acute, death supervening in 3 to 8 days.

**Treatment.**—Rest, and absolute quietness are the first and main considerations. Place in a loose box, with sand, sawdust or chopped straw for bedding; disturb by administration of medicines, feeding, etc., as little as possible; exclude visitors; food to be easily digested, such as sloppy mashes, gruel, linseed tea, milk, green grass if available and given often and in small quantities: strength must be maintained: plenty of cold water for drinking should be always at hand; place watering and feeding utensils on a level with the head. If sufficient aliment cannot be taken by the mouth, quietly feed per rectum. As the disease advances, it is advisable to sling, as by lying or dropping down, symptoms are aggravated, and extension to respiratory muscles is more likely.

Dress wound thoroughly with antiseptics. Strong carbolic acid is the best. Actual cautery is not recommended as it permits the formation of extensive dead tissue which favours the growth of bacilli. Imprisoned dirt, splinters, etc., should be searched for and removed.

Antispasmodics of all kinds have been tried, but with no very satisfactory results. Perhaps the best is chloral hydrate; and it has the further recommendation that it is antiseptic, and given as an enema tends to relaxation of the bowels. Bacelli's treatment with carbolic acid has met with considerable success. One drachm of the pure acid in a 5 or 10 per cent. solution is injected into the neck or shoulders every two hours during the first 32 hours, and less frequently afterwards. As much as 36 drachms may be given in 24 hours as there appears to be a special tolerance for carbolic acid acquired in tetanus. (Winslow.)

The injection of carbolic acid may be alternated with Lugol's solution (iodine and iodide of potassium) either given in drinking water, as rectal enema, or hypodermically.

Tetanus Antitoxin, though useful as a preventive, is little good when symptoms of the disease have developed. No amount of it can withdraw or counteract the toxin in combination with the nerve tissues, although it will neutralise any excess of toxin circulating in the blood stream. It is therefore recommended, that 50 to 100 cc. or even more being given hypodermically or 20 to 30,000 units intravenously as early as possible in the disease. It should be repeated daily or on alternate days, as it is rapidly eliminated.

Thoroughly disinfect the standing on which an affected case has stood, paying particular attention to soil of standing. Anything likely to have

been contaminated by pus from the wound should be burned. Repeat disinfection measures. See " Routine of disinfection " under " General Measures ". Instruments used on infected wounds should not be forgotten.

**Prevention of Tetanus.**—In notorious tetanus districts, use should be made of Tetanus Antitoxin as a preventive in severe wounds, particularly of a deep punctured nature, castration, etc. The dose is 10 to 20 c.c. for larger, and 5 to 10 c.c. for smaller animals. The immunity only lasts three weeks, so that to cover the healing of the majority of wounds, a second injection will be necessary.

**" Tetanus Toxoid".**—A substantial immunity to tetanus can be developed in both animals and man through the administration of tetanus toxoid, a biological agent prepared from cultures of the tetanus organism. The protection given is stated to last for several years.

The toxoid should be given subcutaneously in two doses with an interval of 6 weeks between doses.

## NOTES.

## NOTES.

## TRYPANOSOMIASIS (SURRA).

**Nature.**—" Surra " (signifying " rotten " in the vernacular) is a specific remittent or intermittent fever, due to the presence in the blood of a protozoan organism known as Trypanosoma Evansi, and characterized by a pernicious anæmia, wasting, and in most animals death in varying periods of time.

*Prevalence and susceptibility.*—It is very prevalent in the northern part of India—Punjab, North-West Provinces, United Provinces, Oudh and parts of Bengal, in Rajputana, and in the Bengal Presidency. The whole of British Burma, Assam and Manipur are subject to it. The Deccan is almost immune, and the disease is unknown in the neighbourhood of Madras and the French possessions in the South-East of India. Outside India it also exists on the shores of the Persian Gulf, Mauritius (where a few years ago the disease killed off nearly the whole of animals of the island), French Indo-China, the Dutch East Indies, Yunnan, and the Philippine Islands, Egypt and the Sudan and Palestine.

The disease occurs naturally in horses, mules, donkeys, camels and cattle. It has often been seen as a natural disease in dogs, sporting dogs introduced from England appearing to be particularly susceptible to it: they, moreover, can be most readily inoculated. The disease has also been reported in elephants in Burma.

Though not occurring naturally in them, it can be communicated by inoculation to rats, mice, rabbits, and guinea-pigs, in whom it is invariably fatal.

It does not appear to be transmissible to man. Birds are refractory.

**Horses, mules and donkeys** are most susceptible and frequently in them the disease appears as in epizootic. Death as a rule results, the disease lasting on an average from one to two months, though occasionally it is as short as one week.

**Camels**, irrespective of breed or age, are very susceptible, and the disease is provalent in the localities in which they are bred and live in India. The course of the disease in them if not treated may be as rapid as in equines, but usually it is of a protracted chronic nature lasting about three years, hence the name " Tibarsa " which is given to the disease in camels in some parts of the Punjab.

**Cattle** are much more resistant than the equidæ, and recovery is the rule, though they may pass through a period of extreme emaciation. Cattle and especially buffaloes, can carry the parasite in the blood for a long time without showing symptoms of the disease, so that it is difficult to determine the extent to which it prevails in these animals in this country. The mortality in cattle in Mauritius during a severe outbreak never exceeded 20 to 30 per cent. as against 100 per cent. in horses, mules and donkeys.

The disease has a seasonal prevalence. This corresponds to the rainy season and a month or so after it—that is to say, from the end of June to the end of October. During this period, flies and more particularly biting flies of the Tabanus and Hæmatopota species, abound

which, as will be mentioned later on are concerned in the transmission of the disease. These biting flies have their habitat more particularly in certain areas or zones, *viz.*, low lying marshy lands subject to periodic inundation and subsequent drying up and covered with rough grass and jungle. We have therefore a "Surra Season" and "Surra Zones".

**Protozoology and Infection.**—The Surra micro-organism belongs to a large family of Trypanosomes, now found more or less generally throughout the world, causing disease, of which the following may here be conveniently enumerated:—

(1) Surra, due to Tr. Evansi, in India.

(2) Dourine, due to Tr. Equiperdum, affecting horses and assess in some parts of Europe, Morocco, Algeria, Tripoli, United States of America, Asia Minor, Persia, and in India.

(3) Nagana or Tsetse, fly disease, due to Tr. Brucei, affecting all animals in Tropical Africa.

(4) Mal de Caderas, due to Tr. Equinum, a South American disease of horses and asses.

(5) Gambian horse disease, due to Tr. Dimorphum, affecting horses in the Gambia.

(6) Gall sickness in cattle in the Transvaal, due to Tr. Theileri.

(7) Sleeping sickness in human beings in West and Central Africa, due to Tr. Gambiense, the only trypanosome which so far has been discovered in human beings.

It is particularly mentioned that the first disease producing trypanosomes were discovered by an Army Veterinary Officer, Griffith Evans, in 1880 at Dera Ismail Khan. This was the Surra trypanosome and was named after him.

**Morphology, etc., and how to detect in the blood.**—Surra trypanosomes, in common with other trypanosomes, are parasites belonging to the animal kingdom (not vegetable as in ordinary microbes). They are spindle or fish-shaped organisms, comparatively large, three or four times the size of a red blood corpuscle. They are actively motile, and in fresh blood can be readily seen with a 1/6th power of the microscope wriggling about with great vigour amongst the blood corpuscles. In structure they are bodies of protoplasm, more pointed anteriorly than posteriorly, with a large nucleus centrally placed, and a smaller mass of chromatin, termed the centrosome, at their posterior end. A folded membrane, termed the undulating membrane, commences at the centrosome, runs along the protoplasmic-body, and its thickened border is prolonged anteriorly into a free part constituting the flagellum. It is to the undulating membrane and the free flagellum that the active movements of the parasites are due. With the flagellum foremost they dart like arrows amongst the blood corpuscles, displacing and thrashing them, thus interfering with their functions and causing their death. They do not appear directly to attack the blood corpuscles to absorb them, or penetrate their interior; but for their own nourishment, which

is by osmosis, they abstract nutriment from the blood plasma, probably also depriving the blood corpuscles of hæmoglobin, thus starving the tissues and causing anæmia, wasting and eventually death.

For easy detection in diagnosis, take a drop of blood from a prick or small incision in the ear, place on a slide, cover with cover-glass, do not press the latter down too tightly, and examine with 1/6th power of the microscope at once. A commotion amongst the red blood corpuscles and the presence of the wriggling parasite is very readily seen. They often wriggle off the field of view.

To study their morphology the 1/12th power is necessary. The parasite is easily stained. Leishman's modification of Romanowski's stain is recommended, by whiih method the protoplasm is stained blue, the nucleus violet, and the flagellum and centrosome a bright red The Surra parasite and that of Nagana bear close resemblances.

Trypanosomes are only found in the peripheral blood periodically, usually only for a few days at a time. This should be remembered when examining blood by the microscope.

Multiplication is by division, the centrosome dividing first, then the undulating membrane and flagellum and lastly the protoplasm of the body.

After the death of the animals trypanosomes also quickly die, and blood is not infective after twenty-four hours.

The parasites are very sensitive to heat. Temperatures over 110° F. kill them readily. They are more resistant to cold. Chemical agents destroy them quickly.

Infection is produced through the agency of biting flies. These act as mechanical transmitters, first biting a diseased animal and then conveying the virus to a healthy one. Experiments have shown that in India the genera Tabanus, Hæmatopota (large species) and Stomoxys are principally concerned in the transmission more particularly the Tabanus. Tabanus and Hæmatopota belong to a very large natural order of flies, the Tabanidæ or Horse flies, and may be usually recognised by their large size, and somewhat sombre brown appearance. The blood sucking habit is as a rule confined to the female. The Stomoxys resembles the common house fly and the tsetse-fly and belongs to the same order (Muscidæ).

It has been clearly demonstrated by workers in Africa (Klein, Tante, Bruce, etc.), that all species of trypanosomes capable of being transmitted by the Glossina species of fly undergo a cycle of development in the fly, and that the different groups of trypanosomes may each follow a different life cycle within the body of the fly. Furthermore, tsetse fly, once having become infected remains infective from the time the development of the trypanosome within its body, is complete, up to its death. The same has not been demonstrated to be the case in other species of biting flies, and as stated the transmission of surra through Tabanidæ and Stomoxys, is mechanical.

Cross has incriminated a new genus of tick (Orinthodorus Crossi) as a transmitting agent in the Punjab.

It is well to remember that when surra affected animals are suffering from wounds it is quite possible for ordinary non-biting flies to convey infection from the peripheral blood at such wounds to wounds on unaffected animals.

Three factors are concerned in infection and spread of the disease, *viz.*,—

(*a*) A reservoir of the virus.

(*b*) An inoculator in the shape of a biting fly.

(*c*) A susceptible animal.

Cattle and especially buffaloes, in whom the parasite is carried for a long time without producing symptoms, and camels owing to the length of time they remain alive when diseased, form the reservoirs. It is due to these animals that the disease is carried over from one surra season to another, the surra season being virtually the season of biting flies.

Without these factors there is no disease either in single cases or as an epizootic during the fly season; and conditions of climate, such as heavy monsoon rains with inundation of land, which favour the production of biting flies, also predispose to the incidence of Surra. Tabanidæ require water for their existence and do not live far away from it.

The disease is not produced as was formerly supposed by the ingestion of food or water obtained from low lying marshy and jungly districts, or contaminated by diseased animals; neither is it contracted by eating food contaminated by the fæces of bandicoots or rats which are known to harbour an allied trypanosome (Tr. Lewisi).

Incubation is from 4 to 13 days.

**Symptoms and diagnosis.**—The diagnostic symptom, in all animals, is the presence of the parasite in the blood, found during periods of fever.

**Horses and Mules.**—The invasion of the disease is usually marked by symptoms of a trivial character, those of a slight attack of fever being the only ones noted. Occasionally this fever is accompanied or preceded by a local or general urticarial eruption. In a few days the fever abates, and the animal apparently regains its health. After an interval of a few days (usually one to six days), the animal again becomes ill, temperature is elevated, pulse is full and frequent, 54 to 64 per minute, the conjunctival membrane, especially that of the membrana nictitans becomes the seat of dark red patches of ecchymoses, there is lachrymation, and a slight mucous discharge from the nostrils. At this stage also œdema of the legs, generally from the fetlocks to the hocks, appears. If the blood is examined the trypanosome will be found. Following this second paroxysm of fever, which lasts for varying times, is a period of apyrexia during which symptoms abate, and excepting for the presence of the œdema and a falling off in condition, the animal does not

appear to ail much, and feeds well. A third relapse of paroxysms, however, soon occurs, and all the symptoms are intensified. The action of the heart is irritable, the pulse quick, and the œdema of legs increases considerably. These paroxysms and intermissions continue, the mucous membranes become pale and yellow, anæmia and wasting result, there is gradual loss of strength, and death occurs, usually in one to two months.

The symptoms vary with individual animals but those characteristic of the disease are:—

(*a*) Paroxysms of fever lasting from 2 to 21 days or even more.

(*b*) Intermissions of from 1 to 6 days.

(*c*) Presence of trypanosomes in peripheral blood periodically.

(*d*) Progressive emaciation, in spite of a good appetite, which latter is invariably the rule even during a paroxysm.

(*e*) Ecchymoses of the visible mucous membranes.

(*f*) Extensive œdema of the limbs, sheath and under surface of the abdomen and chest.

(*g*) Progressive and pernicious anæmia.

(*h*) Death.

**Camels.**—In camels the mercuric chloride test has proved to be an easy and efficient diagnostic test. Developed by Bennett in the Sudan it consists in adding one drop of serum from the blood of a camel under test to 1 c.c. of a solution of 1—25,000 of chemically pure mercuric chloride. If the camel is infected a whitish precipitate develops immediately while in healthy camels the fluid remains clear.

In assessing the results of the test the following factors must be considered.

1. An infected camel does not become positive to the test for two or three weeks after infection.

2. A positive reaction develops probably in all infected camels.

3. This reaction does not disappear spontaneously (*i.e.*, if the camel is not treated).

4. The reaction disappears after cure by Naganol or Antrypol.

5. The interval between cure and disappearance of reaction is very variable. It persists for about four weeks in all camels and in some it may last so long as 13 weeks.

6. It is possible that a few non-infected camels may give a positive reaction to the test. The proportion is probably less than 1%.

NOTE.—" The M.C. Test is not a reliable diagnostic test for Trypanosomiasis in Equidæ and Bovidæ."

A mercuric chloride test outfit has been manufactured and is in general use in camel units.

The procedure for carrying out the test on the camels of a troop is as follows:—

Apparatus required.

Three 5 cc. hypodermic syringes complete.

Six spare needles.

150 test tubes (calibre 3/8-in.)

96 Metal clips numbered 1—96. These clips have adjustable collars and can be attached to the necks of the tubes.

6 pipettes to measure 1 cc.

6 hooks for detaching clot from sides of test tubes.

4 metal racks with folding legs. Each rack takes 32 tubes.

3 mops for cleaning test tubes.

12 pipe cleaners for cleaning pipettes.

2 bottles containing mercuric chloride solution.

These are contained in the portable testing outfit.

**Miscellaneous.**

1 dressing tray.

1 set of 4 gallipots.

1 pair scissors.

**Blood collection.**—A troop consists of 96 camels divided into 8 Sections. For convenience in collecting the blood it is convenient to arrange the camels in the form of two squares each side of a square being formed by one section. The table with the apparatus is placed in the middle of the square.

**Control.**—The camel is secured sitting the head in the normal position.

**Site.**—The left jugular vein is raised by pressure of the fingers until the vein stands out very prominently at a point 7 to 8 inches below the angle of the jaw. The site is then prepared and the area cleaned.

**Procedure.**—The needle attached to the syringe is thrust directly into the vein, blood is withdrawn into the barrel and transferred immediately to the test tubes. Do not allow the assistant to relax pressure on the vein, until blood has been drawn into the syringe.

The rack containing the tubes, which are numbered in serial order, is held near the operator. The troop number of the camel has been recorded and check is maintained throughout the test.

When each rack of tubes has been filled it is removed to the storage place immediately, every tube having been stoppered with lint or gauze.

A clean needle and syringe are required for each camel. Boiled water aspirated from a container and expelled into a receptacle is used to clean the syringes.

Detach needles and drop into boiling water before using on next camel. Spare needles are taken into use to avoid delay.

**Staff required**—

1 V. A. S. performing the operation.

4 veterinary dressers, one for tray with clean syringe, one cleaning syringes and one taking over soiled syringes and passing to cleaner.

1 clerk or n. c. o. checking the number of the animals dealt with.

With this staff and procedure a troop can be done in 45 minutes.

**Serum production.**—The racks and test tubes containing the blood are kept in a cool place for 5-6 hours when the clot can be readily detached and either removed or depressed in the tube by the hook contained in the apparatus. The tubes are then allowed to stand over night when the serum clears up. In very cold weather placing the tubes in the sun accelerates clotting.

**The test.**—Test tubes without metal clip are placed in a rack and in each is put 1 cc. of a 1—25,000 solution of chemically pure mercuric chloride. Serum from a serial numbered tube is then taken up by a pipette and one drop of serum allowed to fall into the tube containing the solution. The metal clip is then transferred to the tube containing the mixture and the test read at once.

If the test is positive, *i.e.*, if the camel is affected with trypanosomiasis a definite cloudiness develops almost immediately. A test would be classed as doubtful if a mere haziness developed.

If the test is negative the solution remains clear. The tubes should be examined again after 15 minutes.

It is essential that pipettes are thoroughly cleaned after each test to ensure that serum from one camel does not foul that of another.

NOTE.—It is important that the mercuric chloride solution should be accurately diluted to 1—25,000. This solution can be relied upon for about six weeks under field conditions. After that it should be discarded.

In the camel the attack is usually insidious, the animal presenting no external symptoms, and maintaining its condition for months, though trypanosomes may be swarming in the blood from time to time. Sarwans aver that they are able to diagnose the disease by noting the character of the smell of the urine when the animal is allowed to urinate on a lump of clay. Often the first symptom is a weakness shown when rising under a load. The brightness of the eye, which is so marked in a healthy camel, may be lost, the animal may be dull and listless, and suspicion is thereby aroused. Daily thermal tests with the thermometer for about 10 days may then reveal fever, and the detection of the parasite during the fever stage enables a positive diagnosis to be made. It should be noted that there are enormous differences between morning and evening temperatures in healthy camels: 100° and 101° F. are normal as evening temperatures, but indicate fever at morning time.

There may be pallor of and petechiæ on the mucous membranes, but this is not constant; neither is œdema a constant symptom, though in some cases it is marked, being present in the legs, sheath, scrotum, mammary gland and at the side of the chest, abscesses sometimes forming.

Loss of condition is slow but progressive, the ribs and transverse processes of the lumbar vertebræ stand out, the hump and thighs fall away.

At the commencement of a case, paroxysms are frequent, four or more during a month, but as time goes on they gradually decrease in number, until they may be only very occasional, and short.

Mange is a frequent accompaniment of the disease.

The course of the disease is very variable. Sometimes it is rapid, particularly when the animal is hard worked as on active service, and death occurs in a few months. Usually it is chronic, animals living and remaining in fair condition for years; but death eventually supervenes from anæmia, or from lung complications including œdema of the lungs and bronchopneumonia, which latter are frequently found on *post-mortem* examination.

**Dogs.**—The symptoms of natural surra in dogs may be summed up as follows:—fever, anorexia, œdema of the head and throat, injection of the conjunctiva, corneal opacity leading to total or partial blindness. Trypanosomes are found in the blood during paroxysms.

Surra does not as a rule produce any macroscopic lesions, and there is nothing diagnostic about *post-mortem* examinations. Sometimes there is enlargement of the spleen. The carcase is generally emaciated, and the muscles pallid and bloodless.

### How to deal with an outbreak, or cases.

1. Isolate case, or cases, at least half a mile from unit lines as soon as possible and surround them with a smoke ring to prevent access to biting flies. In a unit where animals are standing close together and facilities exist for inoculation by biting insects, diseased animals are a source of great danger.

2. Remove any doubtful case well away from the lines until diagnosis is certain.

3. As a rule destroy cases, particularly equines when treatment would not appear to be favourable but see remarks under heading of treatment.

4. Bury carcases. They are not infective after 24 hours. Dogs may contract the disease from eating the flesh if they are suffering from wounds in their months.

5. Take temperatures daily up to 14 days (period of incubation) of all animals of an affected unit, and make blood test by microscope of all doubtful cases. Doubtful cases include animals showing fever, debility, and mange (camels).

Be most careful to disinfect properly scissors and instruments used in obtaining blood for examination after each test.

6. Inspect the ground in the vicinity of stables or lines. Any low-lying land or pools within a radius of half a mile, and which are likely to be breeding grounds for biting flies, should receive attention. Lands subject to inundation should be properly drained, and pools treated with kerosene oil and filled in. Tabanidæ never operate far from water.

7. Animals should be clothed (certainly equines) as part protection from biting insects, particularly at evening time from 4 o'clock onwards, and in the morning up to 8-30. Biting flies usually rest in the middle of the day during the great heat.

**During an outbreak,** animals should also be placed in the stable as much as possible, as Tabanidæ usually avoid stables.

8. The use of preventive fly dressings, such as cheer pine oil lightly applied, at the above mentioned times is also of great value.

9. Grooming and cleanliness of the body must not be neglected. A dirty body is always more attractive to flies of all kinds than a clean one. This is well seen in camels, of which grooming is either not performed at all or is perfunctory. Biting flies will always leave a horse for the dirty camel when the two classes of animal are brought together. *Grooming therefore in camels should always be insisted on.*

10. Avoid wounds and their causes, and treat with antiseptics those that occur, covering them up with dressings wherever possible.

11. In the case of camels the lines on which an outbreak should be dealt with are as follows:—

(1) Daily taking of temperatures and examination of blood smears from all camels showing fever.

(2) The immediate application of the M.C. Test to all camels exposed to infection. This will detect cases in the latent period between the attacks of fever.

(3) Retesting after 10—12 days by the M.C. Test of all camels negative to the first test. This test will detect most of the cases which although infected did not react to the first test (*i.e.*, very recent infections).

(4) Retesting about 21 days after the first test of all camels negative to the first and second tests or which have not been diagnosed during the routine clinical examination, mentioned in (1) above.

Unless further new infections are occurring the above should have detected all cases. If further fresh infections are still occurring the application of the M.C. test must be continued together with daily temperature taking and blood examination. Remember that a camel treated with Naganol will probably give a positive reaction to the test up to one month after treatment and possibly longer.

12. **Destruction of camels.**—In view of the very successful results obtained with Naganol and Antrypol only debilitated animals in an advanced stage of the disease should be destroyed.

13. **Treatment in Horses and Mules.**—Various drugs have been tried, most of them being some compound of arsenic and aniline dyes, and meeting with various measures of success, but it was not until Naganol and later Antrypol were produced that a specific treatment was found.

The first trial of Naganol on a large scale in a natural outbreak in this country was carried out at Mona Remount Depot in 1927 and proved an unqualified success, over 90% of animals treated being cured.

The drug was first given by intravenous and intrathecal injections, in accordance with the method initiated and perfected by Dr. Edwards, D.Sc., Director of the Imperial Veterinary Research Institute, Mukteswar.

*Method.*—

A. Make up, just before use, two solutions of the drug, namely (*i*) 10 per cent. and (*ii*) 0·1 per cent. The former is made by taking, say, 5 grammes of the drug, and adding water to dissolve until it reaches 50 cc. The latter is made by taking 1·0 cc. of the 10 per cent. solution and adding water to it until it reaches 100 cc. in bulk. The water should have been boiled beforehand, and strict sterility observed.

B. (*i*) Cast the horse securely, with its head well flexed in towards its chest and firmly fixed with ropes. Clip the region of the poll, and paint with iodine.

(*ii*) A hand's breadth behind the occiput (*i.e.* 3½ to 4 inches) insert the special needle perpendicularly through the mesial axis of the region for a depth of about an inch. Wait awhile and thrust it onwards again a very short distance. Repeat until during a pause, the water-clear cerebro-spinal fluid pours out. Connect up the needle by means of a short length of rubber tubing, with the Record syringe containing the required quantity of the 0·1 per cent. solution of Bayer. After a little practice this operation is done very quickly. The chief points to remember are:—

The needle should be of good quality, with a good sharp point; a blunt needle makes the work impossible; if you encounter bone during the propulsion of the needle, withdraw it some distance and redirect it; if you find much difficulty in finding the correct spot, do not prod, but abandon the attempt until you have studied more carefully the correct relations of the parts.

(*iii*) Inject intrathecally, as above, 20 cc. of the 0·1 per cent. solution per 1,000 lbs. body weight. Simultaneously, inject intravenously 50 cc. of the 10 per cent. solution per 1,000 lbs. body weight (First injection).

(*iv*) A fortnight later, inject intrathecally only 20 cc. of the 0·1 per cent. solution per 1,000 lbs. body weight (Second injection).

(*v*) A fortnight after the second injection, intrathecally 20 cc. of the 0·1 per cent. solution per 1,000 lbs. body weight and 50 cc. of

the 10 per cent. solution per 1,000 lbs. body weight intravenously (Third injection).

Later experience has shown, however, that Naganol given by the intravenous route alone and in reduced doses gives equally good results, and the following method is now advised for use in an outbreak:—

**Curative treatment.**—Two grammes of Naganol or Antrypol in 10% solution given intravenously. On the sixth day after the first injection, a second injection of three grammes in 10% solution given intravenously.

This treatment has been used with success in Assam in treating ponies weighing on an average 500 lbs., and when treating horses or mules of greater weight the dosage should be increased in proportion, *i.e.*, double for 1,000 lbs. animals.

The Naganol or Antrypol should be dissolved in boiled water and filtered through filter paper before use.

Occasionally after curative treatment has been carried out, urticarial eruptions, mild laminitis or cracking of the anus, are observed. These complications pass off in four or five days.

14. **Treatment in bovines.**—Naganol and Antrypol are as much specifics for the treatment of Trypanosomiasis in bovines as in equines. Another drug, however, which gives very satisfactory results is Tartar Emetic, an intravenous injection of this rapidly causes a disappearance of Trypanosomes from the blood. The dose recommended by Edwards is 5 cc. per 100 lbs. body weight of 3·2% solution (*i.e.* fifteen grains to one ounce), repeated every fifth day up to six injections.

15. **Treatment in Camels.**—One intravenous injection of two grammes of Naganol or Antrypol is sufficient to cure the majority of cases but where the M. C. test applied after four weeks or microscopic examination of blood films show that the first dose has been ineffective a second dose of four grammes should be given.

16. **Treatment in dogs.**—There is very little literature available on the treatment of Surra in dogs, but of the drugs that have been tried Naganol or Antrypol would seem to offer the best hope of successful treatment. Early treatment is essential, in order that the parasites may be destroyed whilst they are confined to the blood circulation before they have invaded the cerebrospinal system. The dose recommended by Edwards (Director of the Imperial Veterinary Research Institute) for a large dog (Fox-hound) is half a gramme (5 cc. of a 10 per cent. sol.). The drug is given intravenously and is repeated after an interval of 3 weeks to one month. This dosage is well tolerated and was very successful when employed by Lt.-Col. E. C. Webb, R.A.V.C., in an outbreak of Surra amongst the Delhi

Hounds during the season 1927-28. In this outbreak 10 hounds were diagnosed positively as Surra at the beginning of February 1928. The whole pack was immediately treated with a curative dose of Naganol and the injection was repeated after an interval of 3 weeks. Immediately after the first injection the affected hounds appeared to be cured and from this time to the end of the season continued to hunt regularly, nor did a single hound in the pack miss a day's hunting on account of Surra after the first injections were completed. A report five months later from the hills whither the pack had been sent to summer, stated that all hounds were fit and well. Edwards further states that Naganol given in smaller doses at short intervals (once a fortnight) acts as a preventive to Surra infection. This line of preventive treatment might therefore be adopted in dealing with a pack of hounds in a district in which there was grave danger of Surra infection.

**Prevention.**—With regard to the camel the mercuric chloride test enabling early diagnosis to be made has simplified the control of surra among these animals enormously and as in Naganol and Antrypol we have a simple and easily administered curative treatment, surra has lost a great deal of its terror. In horses and mules no successful diagnostic agent has yet been discovered; however, Naganol given intravenously has prevented animals on military operations in surra zones becoming affected and similar doses have been used with success in preventing the spread of the disease when it has appeared.

As however it is desirable to use all means to prevent infection the following considerations should be given due weight:—

1. No serum preventive inoculation has up to the present been proved to be of any use.

2. Avoid known surra zones during the surra season as much as possible.

3. If this cannot be done, make double marches through extensively irrigated districts, and night marches through narrow belts of irrigation or across belts of land subject to inundation from rivers and *nallahs*.

4. Form camps on high lying grounds, and at least half a mile from standing water.

5. The use of light jhools, whenever possible, is recommended, particularly for horses and mules, when marching through known surra zones during the season, and preventive fly dressings should be resorted to.

6. Concentration or parades of camels should not ordinarily be made in irrigated and river districts or at the foot of hills (all surra zones) before 15th October.

7. For grazing during the surra season, camels should, where circumstances admit, be removed to high dry lands, away from rivers

and inundated lands. If the latter must be grazed on, animals should graze during the middle of the day only, and be placed on land at least ½ a mile away from irrigation for the rest of the day.

8. Camels should not be purchased between 1st July and 31st October, as so many surra affected animals are in good condition during that time.

The disinfection of the instruments used for obtaining blood after each test is again mentioned. This is best effected by passing through a flame.

9. Cleanliness and grooming of camels should be enforced.

10. The avoidance and care of wounds should be impressed on all Sarwans.

NOTES.

## TRYPANOSOMIASIS (DOURINE).

**Synonyms.**—Maladie du Coit, Venereal Disease of the Horse, Equine Syphilis, Breeding Paralysis. Dourine (Arabic signifies dirt).

**Nature.**—Dourine is a contagious venereal disease affecting equines, due to a trypanosome (Trypanosoma Equiperdum), transmitted chiefly by coitus, and characterized first by local swellings of the genital organs with discharge from the penis or vulva, afterwards by exanthematous patches or, "plaques" on different parts of the skin, progressive anæmia and emaciation, and lastly Paralysis and death. The disease is usually of a chronic nature.

**Prevalence.**—So far as is known, the disease does not now exist in India. Previous to 1902 it had in all probability been existent for a considerable period of time, but in that year a widespread epizootic of the disease came to light, especially in the Punjab and in certain districts of United Provinces. The true recognition of its nature, its cause, and the prompt measures taken in respect of it, have, however, led to its extinction. Being a venereal disease and therefore relating to breeding operations, and the horse and mule breeding operations in India being for the most part under Government control, suppression of the disease comes all the more easy.

With regard to countries from which animals are imported into India, it may be mentioned that England, Australia, South Africa, Argentina and Arabia proper (so far as is known) are free. Syria and Persia appear to be infected to a limited extent.

**Susceptibility.**—Practically it is a disease limited to horses and donkeys and only those used for breeding purposes are affected naturally by it. Stallions and mares are very susceptible, donkeys much less so. Geldings and mules are susceptible to experimental or accidental inoculation.

Dogs do not suffer naturally but they are susceptible to inoculation, and sometimes they are made use of for diagnostic purposes, especially in the donkey, in which animal diagnosis is often difficult. Pariah dogs are very resistant.

Rabbits, rats and mice are infected by inoculation, and may carry on the disease by coitus.

Cattle are considered immune, except to over-powering doses of infective blood, and even then the trypanosomes do not remain for very long in the blood.

No cases have ever been reported in man. Human syphilis has no connection with the disease.

**Protozoology and infection.**—The Trypanosoma Equiperdum resembles that of Surra and Nagana, but is slightly smaller, being 18 $\mu$ to 26 $\mu$ long. It is considerably less active than the trypansoma of Surra and it is never so numerous as that parasite. It is found in the semen and discharge from the penis of the stallion, and the mucous discharge from the vulva of the mare. It is also found in blood taken from the œdematous swellings and plaques, but more rarely in blood from other regions. The best time to find the trypanosome

in the plaques is on the first appearance of the latter: some hours afterwards, they are difficult to find in these lesions. They persist for a longer time in the œdematous swellings. The fluid from these swellings may not contain them, and it is necessary to draw blood for their detection. As they are never very numerous, the smear on the slide for microscopical examination should be a large one extending over the most of the slide, so as to give a large field to work over. The parasite retains its motility only for a few hours outside the body. It is advisable to fix and stain, Leishman's stain being very suitable for the purpose.

**Infection.**—The natural mode of infection is by coitus. The virus thus differs from other trypanosomes in that it has the power of penetrating intact mucous membranes. Infection apart from coitus is extremely rare, but infection through the medium of grooming utensils such as sponges, or by means of contaminated litter, is quite possible. It is very doubtful if it is spread naturally by insects, although stomoxys calcitrans has been proved to transmit the disease experimentally.

Blood drawn from an affected animal is not virulent after twenty-four hours, and presumably discharges from the penis and vulva also quickly lose their virulence.

Two-thirds of the mares served by an affected stallion contract the disease.

## Symptoms and diagnosis.

Incubation of the disease contracted by coitus is from eleven to twenty days.

Symptoms may be divided into three stages, *viz.*, (*a*) primary, (*b*) secondary, and (*c*) tertiary.

(*a*) **Primary stage.**—In the stallion the onset is so insidious that several "coverings" may have taken place before danger is apprehended. The first noticeable sign is a little œdema of the lower part of the sheath, which may at first be overlooked. Examination shews œdema of the penis and discharge from the meatus urinarius, with redness and eversion of the mucous membrane of the urethra at that part. The swelling of the sheath gradually extends to the scrotum and inguinal region, and the under-surface of the abdomen. The swelling is usually cold and painless but, sometimes may be hot and tender. On account of the irritation there are frequent erections, and the desire to cover mares is increased; but though the animal is at this stage capable of performing the act, he is entirely sterile. Later on, erosions and ulcerations may appear on the penis and scrotum.

In the mare the symptoms are less marked. They consist at first of a unilateral or bilateral swelling of the vulva often extending up to the anus, a bright red colour of the mucous membrane of the vagina, and a viscid discharge. Micturition is frequent.

The temperature at this stage may be slightly increased (101·4 or so), and the appetite remains good.

(*b*) **Secondary stage.**—After a period of four to six weeks, round or oval eruptions, varying in size from a rupee piece to the size of one's hand, appear on different parts of the body, usually about the neck, shoulders, fore part of the chest and back but sometimes also on the loins, quarters and thighs. These eruptions are termed "plaques", and are pathognomonic of the disease. They are salient, may be felt by passing the hand over the body, and look as if a metal disc had been placed under the skin. Sometimes they are so slight that it is only by looking sideways along an animal that they can be detected. Their duration is very variable; they may appear in the morning and disappear the same night without a trace, or they may persist for five to eight days. Sometimes they become œdematous and persist for a slightly longer period. They are not at all hot and painful. Occasionally the plaques are preceded by an evanescent urticaria.

Anæmia and wasting are now well marked. The animal is listless, constantly lying down, and experiences difficulty in rising. Symptoms of paralysis are noticeable, the animal dragging its hind feet, or knuckling over at the fetlock when being walked. There is also tenderness of the loins on pressure. The swellings at the genitals are hard and chronic; and lymphatic glands, especially the inguinal, shew enlargement and may suppurate. The appetite still remains good. Fever is present, but is intermittent, and never high (102° F.). Connection is practically impossible in the stallion; and mares, if they conceive, generally abort.

(*c*) **Tertiary stage.**—This is characterized by rapid progressive anæmia, loss of co-ordination, paralysis and death. Recovery is rare in stallions; 20 to 30 per cent. of mares recover.

The total duration of the disease in India is from twelve to eighteen months.

The symptoms in donkeys are very similar to the horse but the disease is slower and more chronic, and difficult to detect in the early stage. It is frequently only recognised after a large number of animals have been infected.

**Differential diagnosis.**—The disease in its early stage may be mistaken for Surra, but the history of the case will enable us to differentiate. Dourine does not attack geldings, and mares that have not been to the stud. The parasite of Surra is found teeming in the peripheral blood when the temperature is high. The temperature in Surra shows a very high rise, and a corresponding low fall. In Dourine the temperature is never high, and the parasite is not found or with difficulty found in the peripheral circulation. Once plaques appear, differentiation is easy.

Purpura hæmorrhagica in the early stages may have some likeness, but the history and character of the swellings of Dourine are different.

The disease in some respects may simulate Glanders Farcy, but the latter is distinguished by the Mallein test. Vesicular Exanthema may also complicate Dourine. The former is a benign disease, manifesting itself from one to six days after "covering" by heat and swelling of the genitals with subsequent formation of vesicles, pustules and scabs. The disease passes off in three weeks, leaving white patches (leucoderma) on the tissues of the external genital organs.

**How to deal with an outbreak, or cases.**

1. On the reappearance of the disease in India, the policy to be adopted is one of "stamping out" by destruction of all affected cases, mares and stallions. This can readily be effected owing to horse and mule breeding being under Government control as before mentioned.

2. Should there be re-occurrence, the following measures would be necessary in districts where the disease prevails:—

(1) Carefully inspect all mares about to be put to the horse. Refuse all mares that have a discharge from the vulva other than ordinary manifestation of œstrum, or shewing any swelling of that part. Refuse mares shewing suspicious œdematous swellings in any other region; also, old and weakly mares.

(2) Examine the stallion's penis frequently and carefully. If there is the slightest lesion on it, he must not be used until all doubt as to its nature has passed away. Special care should be exercised in the periodic examination of jack donkeys employed for mule breeding.

(3) Circulate all information possible to breeders and owners of mares as to the nature of the disease.

(4) Enforce immediate reporting of the disease in both stallions and mares.

(5) Interdict the sale or removal of mares from an infected district.

(6) If the malady has spread in a district stop the use of all stallions the property of Government or private property.

(7) Destroy all affected animals; assess value for compensation. Treatment is of little avail; the disease is always protracted, and considering the small percentage of recoveries it is unwise to resort to it. In countries where it is practised, it is usual to castrate affected stallions and to brand affected mares with a large D to exclude them for breeding purposes.

NOTES.

# TUBERCULOSIS.

**Synonyms.**—Consumption, Tabes, Scrofula, Pining, Grapes, Phthisis. This disease is not frequent in India, and is rarely seen amongst animals used in military service. This may be due in part to an inhibitory effect of a tropical climate on the germs, the disease chiefly occuring in temperate zones; but more perhaps to the practically open air existence of animals lessening the chances of infection. The history of tuberculosis throughout the world shows that prevalence bears relation to the opportunity for infection rather than climate, latitude or altitude, animals in the open practically being free, whilst animals in confined byres, etc., like the cows of temperate or colder climes suffer.*

The adoption of military dairy farms in India and the herding of large numbers of animals together in consequence, however, makes a consideration of the disease necessary.

**Nature.**—Tuberculosis is an infectious, incurable disease, common to man and a large number of animals, caused by the Bacillus Tuberculosis, and characterised by the formation of tubercles in the lungs and other parts of the body, with a tendency to necrotic degeneration, caseation and later calcification. It is usually of a protracted nature.

**Susceptible animals.**—Nearly all animals can contract the disease, either naturally or by inoculation.

Cattle are very subject to it. Sheep and goats under usual conditions show a remarkable immunity. Horses, asses and mules are rarely affected. Pigs contract it readily, mainly by ingestion. Dogs and cats rarely show the disease naturally, but contract it readily on inoculation. Apes and monkeys in confinement almost all die from the disease. It has been definitely demonstrated in camels in India. It is very frequent in birds, often occurring as an epizootic amongst barn door fowls, hens, turkeys, and ducks. It is common in pheasants and pigeons. Cagebirds, *e.g.*, parrots and canaries, all suffer. Guinea pigs show a stronger susceptibility to the human and bovine forms of the disease, and rabbits to the avian form on inoculation. Lastly the human being is very susceptible to the disease.

**Bacteriology and infection.**—Three forms of the disease are now recognised viz., the human, bovine and avian forms. The causal agent, the Bacillus Tuberculosis, in each of these is primarily and essentially the same, but in different hosts and environments it takes on different habits, and its pathogenic attitude towards different races becomes modified. So modified does this become, that it is often with difficulty that it can be transferred from one genus to another. Thus bovines show a limited receptivity for the human bacillus, and the bovine

* Note.—Investigations of recent years into the incidence of tuberculosis in India have shewn that the disease is far more prevalent than was formerly supposed. In slaughter houses under veterinary supervision it is not uncommon to find organs or glands with tuberculous lesions, but cases of advanced tuberculosis with extensive involvement of the pleura and thoracic lymphatic glands are rarely if ever seen.

bacillus has a greater potency over that of man in its action on pigs and small rodents. As a proof of interchangeability, however, the frequency of tuberculosis in human beings and cattle in the same country or district, or the tuberculosed parrot or canary or the consumptive human being, may be cited. So frequent is this that it may safely be set down as a rule.

The Bacillus Tuberculosis is rod shaped with rounded ends often slightly curved 1·5 to 3·5 μ long, 2 μ thick about three quarters the diameter of a blood corpuscle, shorter and thicker in the ox, usually occurring singly but two and exceptionally three or four together may be noted. It is non-motile, and very slow in its growth both in the animal body and on culture media. It is an obligatory parasite though there is every reason to believe that it can live as a saprophyte. It has a remarkable staining reaction, retaining the red colour of fuchsin after treament with strong mineral acids or absolute alcohol which rapidly decolorize most other organisms. It is therefore termed "acid alcohol fast". This reaction is made use of for diagnostic purposes.

It grows equally well in the presence or absence of oxygen, but is prejudicially affected by light. Diffused daylight will kill it in culture in 7 to 18 days. Strong sunlight kills it in sputum on a solid surface in several hours: mixed with soil it survives 137 days. In dried sputumn it may remain infective for 9 or 10 months, and in water it is alive after 50 to 70 days. Moist heat at 140° F. sterilizes in one hour: boiling kills in half an hour. Freezing does not sterilize. Salting kills it in 1 month. 5 per cent. solution of carbolic acid kills it in 30 seconds, and 1 in 1000 mercuric chloride destroys it in 10 minutes.

Infection is by inhalation, ingestion and inoculation. In cattle the first channel is the usual, a diseased animal infecting others in a stable or standings, the microbe being forcibly expelled into the air from diseased lungs by coughing.

Drinking tuberculous milk is also a frequent cause in young animals, the digestive system being first affected. Human beings are similarly infected. In addition, tubercular warty growths in the skin from inoculation are frequently seen (Lupus).

Pigs usually contract the disease from ingestion, the lesions being usually found in the pharynx, intestines and mesentery. Rats and mice are also affected in this way.

It is very rare that human beings or animals are born with tuberculosis. The disease as such is not hereditary, and animals, born of diseased parents can be reared without contracting the disease. There is, however, a racial vulnerability. Other predisposing causes are:—heavy milking in cows, breeding too young, inbreeding debilitating disease, overcrowding, etc.

**Symptoms and Diagnosis in Cattle.**—It is usually of a chronic nature, and shews itself chiefly in pulmonary, abdominal and mam-

mary forms. It may last for months without suspicion. In the pulmonary form the lungs, pleura and lymhatic glands of the thorax become affected. There is cough, blowing, areas of dullness and crepitation or wheezing in parts on ausculation of the lungs, the animal stands upright and evinces pain on pressure over the ribs. Later on these symptoms are more pronounced, and the temperature, which was inappreciable at first, becomes higher. Loss of condition and an unthrifty appearance are now much in evidence. In later stages this becomes a wasting to thinness, cough is more frequent and painful, and mucous membranes are anæmic. There is then no mistaking the disease. In the abdominal form, which has been termed Tabes Mesenterica, the intestines, mesenteric glands, peritoneum, liver, spleen and pancreas become affected. There is a steady loss of condition in spite of good feeding, and digestive trouble, such as tympany, irregular bowels, etc., set in. Tubercles may be felt on the rumen or mesentery per rectum. Animals with abdominal tuberculosis are known as "Piners". Mammary tuberculosis may be primary from external infection of milk ducts, but more commonly is secondary to a general tuberculosis. At first rather a firm uniform painless swelling of one or two quarters of the udder (usually the hind ones), without serious interference with the milk secretion, is noticed. Afterwards an irregular knotted condition of the gland is developed, and the milk becomes pale, watery, semicoagulated and filled with bacilli. The climax is reached in a dense induration of the gland. The mammary lymphatic glands, behind and occasionally in front of the udder, become swollen, and finally indurated, with caseation.

Symptoms in other animals are more or less the same as the above described for cattle varying according to seat and manner of infection.

*Post-mortem* appearances are characteristic of the disease. These consist in the formation of tubercles or nodules in the lungs, pleura, bronchial and mediastinal glands in the pulmonary forms, and tubercles in the intestines, mesenteric glands, liver and spleen in the abdominal form. Tubercles may be found in almost every organ or tissue. A tubercle takes its name from a small rounded nodule, which at first virtually invisible, increases, to the size of a pin's head or a millet seed (miliary tubercle) and which by confluence with others, form conglomerate masses of all sizes varying from the size of a pea or nut upwards, also termed tubercles. The latter, on the pleura and peritoneum, form pale red cauliflower-like growths spoken of as "grapes".

The miliary tubercle is greyish and translucent, and is the seat of action of the bacillus. It consists of a large cell or cells termed "giant cells" in the centre, with a zone of large epitheloid cells and a second or outer zone of small round lymphoid, cells, surrounding. The bacilli are found in the giant cells. The tissue round the miliary tubercle is red and congested.

After a time the miliary tubercles, and particularly the larger tubercles, degenerate in the centre into cheesy matter. This caseation is a prominent feature of tuberculosis, and is almost pathognomonic of the disease. In old cases the tubercle may be calcified, grating when cut with the knife. This is especially so in cattle. From secondary infection by pus germs, abscesses may be observed.

**Diagnosis** may be confirmed by the miscroscope or by the Tuberculin Test.

The use of the microscope in diagnosis is not altogether satisfactory in veterinary practice as expectorate in animals is always swallowed. In cases of suspected tuberculosis of the udder, the milk, however, can be examined by the microscope. Special staining is necessary. The best method is Ziehl-Neelsen's carbolate of fuchsin (made by a 5 per cent. solution of carbolic acid and 1/10th of its volume of fuchsin). Wash off superfluous stain with water and decolorize with a 5 per cent. solution of sulphuric acid or a 15 per cent. solution of nitric acid. When decolorized, rinse well in 70 per cent. alcohol and afterwards water. Next counterstain with 1 per cent. aqueous solution of methylene blue for one minute, washing off excess of stain with water. The bacillus of tuberculosis is stained red, all other bacteria blue.

**Tuberculin Test.**—Tuberculin, which is the sterilized and filtered product of the growth of the Bacillus Tuberculosis in bouillon, has the property of causing a reaction in a tuberculous animal. The test when applied subcutaneously consists of a steady rise of temperature of 2° F. or more in the course of the next twenty-four hours after injection of a dose, the rise being usually between the 8th and 16th hours, and an equally steady subsidence to the normal.

**Procedure of test.**—Carefully take the temperature of the animal previous to testing. Test at 10 or 11 P.M. Carefully disinfect syringe first by boiling or by solution of acid carbolic. Dip needle of syringe into strong carbolic acid before inserting it into the skin, and on withdrawing it. Take temperature at 6 or 7 A.M. eight hours after injection, and every 2 hours till the 16th hour. Record on a temperature chart so that the gradual rise and fall of a typical reaction may be readily seen. The precautions to be observed are that there should be no fever before testing, that animals should not be exposed to the hot sun or sudden variations of temperature but living under their ordinary conditions. Cows should not be within three weeks of parturition, or within three days of œstrum. Rest and gentle handling are essential.

Other methods of applying the Tuberculin Test are:—

**The Ophthalmic or Conjunctival method.**—In this method a few drops of specially prepared concentrated tuberculin are dropped into the conjunctival sac, the head being held up until the tuberculin has become distributed under the lids. In a tuberculous animal there

is a reaction in the tested eye, consisting of a temporary conjunctivitis with a collection of purulent secretion at the inner canthus. The reaction may commence at the 8th or 9th hour and may last for 20 hours or several days. One application of this test does not render a subsequent application useless; in fact the test can be repeated in a few days. Furthermore, a previous application of the subcutaneous test does not interfere with this test. The opthalmic test can be carried out simultaneously with the subcutaneous test.

**Intra-dermic method.**—In this method 4 or 5 drops of specially concentrated tuberculin are injected into one of the folds of the skin under the base of the tail. A positive reaction consists of œdema and swelling of the injected fold up to the size of a Brazil Nut or larger in about 24 hours, persisting for several days. The opposite fold undergoes no change.

**The Double intra-dermic method.**—This method was recently the subject of investigation of a Special Committee of the Medical Research Council which was appointed to test the reliability of the various methods of tuberculin testing. This method has been tested as to its efficacy in Indian cattle at the Imperial Veterinary Research Institute, Mukteswar, and the following instructions for carrying out the test have been issued by the above-named Institute.

The test is performed by injecting a small quantity (1/10 cc.) of undiluted tuberculin of known potency and purity *into* the skin, preferably of the side of the neck, in two successive doses. The second or test dose is injected 48 hours after the first or sensitising dose—observations of the resulting reaction are made by palpation and measurement of the swelling produced at the site of inoculation.

A.—Technique.

(1) Shave and cleanse an area of skin, about 4 square inches in extent, at the middle of the side of the neck. This is done preferably on the day preceding the first injection.

(2) Pinch up a fold of the shaved area and hold it firmly between the thumb and forefinger of the left hand.

(3) Insert obliquely into the fold of the skin the needle of the hypodermic syringe, containing the requisite quantity of tuberculin. (The needle should be short and strong, and the syringe carefully tested beforehand to ensure absence of leaking when pressure is applied to the piston; an all-metal dental hypodermic syringe has been recommended; the syringe and needle should be sterilised by boiling.) The depth to which the needle is inserted into the skin is of importance. It will vary somewhat with the thickness of the skin. The reaction produced after a deep injection, made *into* the dermis, is more marked than that observed after an injection made superficially, just under the epidermis. On the other hand, take care to see that the needle does not pass too deeply into the subcutaneous tissue. When the needle has been correctly inserted, inject the tuberculin into the dermis, for which operation the application

of considerable pressure may be necessary. The proper injection of the tuberculin becomes evident immediately by the appearance of a pea-like nodule in the skin.

(4) Inject the second, or test dose into the skin after an interval of 48 hours from the time of the first injection. The dose is the same as the first dose (1/10th cc.). The interval may, however, be prolonged to 72 hours. Some degree of swelling is always produced after the first injection, even in animals that are not tuberculous, but the swelling is much greater in animals that are tuberculous. For the second injection therefore, inject the dose of tuberculin, in exactly the same manner as the first, into the centre of the swelling produced by the first injection.

B.—Observations.

(5) Record by actual measurement the thickness of the fold of skin at the seat of injection. The measurement should be made by means of a pair of callipers, the jaws of which just grasp the injected fold of skin. The distance between the jaws is then read on a ruler.

(6) Measure the thickness of the fold immediately before the first injection, and again at each interval of 24 hours afterwards. Measure also in this way the thickness of the fold immediately before the second injection. The initial measurement gives information concerning the normal thickness of the skin and serves also as a guide to the depth at which the injection must be made. Take care to exert an uniform degree of pressure on the skin with the callipers at successive readings so as to obtain comparable readings.

(7) Palpate also the swelling at the seat of injection, and record the presence or absence of heat, tenderness, and œdema, and the consistence of the swelling.

C.—Estimation of Result.

(8) In animals that are not tuberculous a small swelling is produced after the first injection; the skin at the seat of injection is found to have increased in thickness on measurement 24 hours after the injection, but the increase does not usually exceed a few millimeters; after 48 hours, the skin shows some decrease in thickness. Frequently, no change in thickness is observed. Palpation reveals absence of local heat and tenderness, and a pea-like nodule is often felt, which is not surrounded by an œdematons infiltration.

(9) In animals that are tuberculous there is a considerable increase in thickness of the fold when it is measured 24 hours after the first injection. The swelling persists, and often increases, till the 48th hour reading; sometimes, however, there is a slight decrease at the latter reading. Palpation reveals an appreciable degree of local heat and tenderness, with a variable degree of surrounding œdematous infiltration; these characteristics give the swelling a peculiar sensation to the touch, which is different from that felt on palpating the skin of the animal which is not tuberculous.

(10) The most decisive information is given by examination of the skin 24 hours after the second injection.

In animals that are not tuberculous, the skin shows very little increase in thickness; and on palpation, very little œdematous infiltration.

In animals that are tuberculous, however, the swelling and surrounding œdema are considerably increased as a rule; the swelling may reach a thickness of 50 millimeters or even more.

In some animals that are tuberculous, the reaction may not be very striking at this stage. In such cases, where doubtful reactions are observed, a reading made 48 hours after the second injection reveals the swelling to be still distinct, whereas in an animal that is not tuberculous it will have subsided.

(11) Although actual measurements serve as a useful means of establishing permanent records of the test for subsequent reference, the presence or absence of a reaction is determined mainly by observing the characters of the swelling as revealed by palpation of the seat of injection, and this applies more especially to tests in which the swelling is not very marked.

(12) The reactions observed in buffaloes are, as a rule, more severe than those observed in cattle; sometimes the œdematous infiltration is so great that it is difficult to pinch up a fold of skin for measurement at the site.

(13) A control test may be carried out by injecting 1/10 c.c. Sterile broth or normal saline on the opposite side of the neck at the same time as the tuberculin is injected. This may help in the estimation of results in doubtful cases.

## How to deal with the disease.

1. Destroy all diagnosed cases, particularly, if there is emaciation, or tubercular disease of the udder in cows.

2. In dairy herds if there is any doubt, test with Tuberculin, and destroy reactors.

3. Imported animals should undergo the tuberculin test before purchase, or before admission to dairy herds.

4. Thoroughly disinfect the standings of affected animals in accordance with instructions laid down in "Routine of disinfection". Pay particular attention to mangers, walls, flooring, bedding or anything contaminated from exhalation, from probable discharge from nose or mouth, and excrement. The latter should be burned, as the fæces are likely to be infective owing to animals swallowing expectorate from their lungs; the bacillus is not killed by the gastric juice.

5. Do not use milk from cows with indurated or diseased udders, until it can be proved that they are not tubercular.

6. When from loss of condition or chronic wasting an animal is considered suspicious of tuberculosis, boil the milk for ½ an hour

until the tuberculin tests shows that the wasting is not from tuberculosis.

7. Milk vessels should be thoroughly cleansed. Steam and boiling water are best for this, as medicinal agents taint or spoil the butter and milk.

8. The flesh of carcases in the early stage is fit for food if properly cooked, and any intermuscular bands removed. Internal organs and lesions on the costal pleura and peritoneum should be destroyed by fire. Meat is less virulent than milk because muscle is not usually a seat of the disease and because it is usually cooked before being eaten. Except in generalised tuberculosis, the blood is not permanently infected. Animals showing extensive disease on slaughter, or emaciated animals with advanced tubercular lesions, should not be used as food.

# NOTES.

# NOTES.

## ULCERATIVE LYMPHANGITIS.

**Synonym.**—Ulcerative cellulitis.

**Nature of Disease.**—A contagious disease characterised by inflammation of subcutaneous lymphatic vessels resulting in abscess formation and ulceration.

**Susceptibility.**—Horses, asses and mules are susceptible, the large majority of cases being seen in horses.

**Prevalence.**—The disease may occur in any country and is particularly common in France. Under ordinary peace conditions when animals are kept under good hygienic surroundings its incidence is much reduced. Before the Great War it was practically non-existent in Great Britain and in India. For several years after the Great War a number of cases occurred from time to time amongst military animals in England and in India, but these cases were the aftermath of the war, and the majority of them were not fresh infections but old chronic cases which had returned from the seat of war, apparently cured, and had broken out afresh.

In France and Flanders during the Great War this disease was one of the biggest causes of inefficiency amongst horses. Advanced cases were practically incurable, or at any rate the length of time required to effect a cure was so protracted that their retention was not an economical proposition. Fortunately the lesions are more or less localised being confined to the limbs, and for this reason the Army was able to dispose of all such incurable cases to the butchers in Paris at a very reasonable price, for human consumption. Thousands of animals were so disposed of, and had it not been for this method of disposal the financial loss would have been enormous.

**Bacteriology and Infection.**—The causal agent is the Preisz-Nocard bacillus. It gains entrance through wounds or abrasions of the extremities from infective soil. It may also be conveyed from animal to animal by contaminated grooming utensils, litter, clothing, men's hands, etc. Unhygienic surroundings favour the incidence and spread of the disease. Any circumstances which may have a debilitating effect upon the animal's tissues will favour infection. Hence, active service conditions, where animals are picketed in the open on muddy standings, and exposed to inclement weather are ideal for infection and spread of this disease. Furthermore, animals picketed in the open are much more liable to wounds and abrasions of their extremities from kicks and shackle chafes, and such wounds and abrasions are apt to be overlooked; another contributing factor is that, even if not overlooked, the circumstances may be such that dressings become almost immediately soiled.

**Symptoms and Diagnosis.**—The disease most commonly affects the hind legs. The first symptom to be noticed is a certain amount of swelling, which may be localised at the spot where the abcess is forming or may be more or less general, affecting the lower half of the lim. Lesions are common anywhere between the hock and

fetlock, including these joints. The swelling points at some spot and bursts, discharging at first a blood streaked, thin, yellowish discharge, which later becomes thicker. The abscesses, before bursting, are bud-like in appearance, varying in size from a bean to a walnut. The formation of these buds on a locally inflamed and swollen area, or on what looks like a limb affected with lymphangitis is characteristic, and in the majority of cases can be clinically differentiated from the buds of Epizootic Lymphangitis. In this latter disease the buds may occur anywhere in the neighbourhood of a wound or scar, and although lymphatics in their neighbourhood may be inflamed and corded, there is rarely any general swelling of an inflamatory nature; also the buds in Epizootic Lymphangitis are more sluggish, and if opened, are found to contain a thick creamy pus, which is not retained under tension. Moreover the subcutaneous tissue is not involved to the same extent as in Ulcerative Lymphangitis. In Ulcerative Lymphangitis the bud after bursting become converted into granulating ulcers. If small, and there is little involment of the subcutaneous tissues, healing may be rapid, but in the meantime other buds may have formed and burst. A mild case may be cured within a few days, or a week or two, but in more severe cases the ulcer, instead of healing, eats deeper and deeper into the subcutaneous tissues, until large areas, frequently in the neighbourhood of the back tendons are involved. In this latter case it may be impossible, even with surgical interference, to reach all the diseased parts. These cases may go on for months if treatment is persisted in but in many such cases it is more economical to destroy the animal. The opinion of some veterinary officers is that the less one interferes surgically with the abscesses, after they have burst, the better, and that extensive surgical interference favours extension of the lesions. Certainly, small lesions will heal rapidly with ordinary simple treatment, but deep seated lesions with or without surgical interference are very troublesome. One of the worst features of the disease is its tendency to recurrence. Lesions may heal quickly or slowly; and the animals be discharged to duty, but in a large number of cases the same animal is admitted at a later date with a recurrence of lesions. It would appear that the organism may lie dormant in the tissues after it has once gained a footing, to renew its activities from time to time. Superficial lymphatic glands may be involved, becoming swollen and prominent, but they never suppurate. Lameness only occurs when lesions are in the neighbourhood of joints or tendons, and tendon sheaths are frequently involved in the ulcerative process. At the commencement of the disease there may be some slight constitutional disturbance, but as a rule animals are not affected in this way and remain in good condition.

Lesions may occur in situations other than the limbs, but are very rare. Lesions have also been reported, as occurring internally in the lungs and kidneys. It would seem that the heavier and coarser bred animals, those of the so-called lymphatic type, which are more

subject to ordinary lymphangitis, are also more susceptible to this disease. The disease was particularly prevalent amongst heavy draught and light draught horses in France.

**Differential Diagnosis.**—There are two diseases with which Ulcerative Lymphangitis might be confounded, *viz.*, Glanders and Epizootic Lymphangitis. If there is any doubt the Mallein Test should always be applied. Epizootic Lymphangitis is very readily diagnosed by the microscopical examination of pus from a freshly opened bud. This pus will be swarming with cryptococci.

**Treatment.**—Mild cases of the disease require no special treatment. The abscess cavities may require opening up and possibly slight curetting, after which healing may occur rapidly under simple antiseptic dressings.

Vaccination should offer the best hope of effecting a permanent cure. This method of treatment was tried to a limited extent in France during the Great War, but the results were not altogether convincing.

Routine disinfection should be carried out. All cases should be strictly isolated and special care taken in their dressing and more particularly in the disposal of soiled dressings. Anything that can become contaminated with discharges should receive special attention and the floors of stables and dressing sheds should be regularly and frequently disinfected. Preventive treatment consists in daily careful inspection of all animals in a unit for the purpose of detecting fresh wounds and abrasions. Every effort should be made to get animals on to dry standings, and all measures which will tend to prevent kicks, galls and injuries of all sorts will be measures directed against the incidence of this disease.

NOTES.

# VARIOLA.

## (Horse-pox—Cow-pox—Sheep-pox—Camel-pox.)

Under the generic name of Variola, the following diseases have been grouped:—

Small-pox in human beings (Variola).
Cow-pox (Variola Vaccinæ).
Horse-pox (Variola Equinæ).
Sheep-pox (Variola ovinæ).
Goat-pox (Variola caprinæ).
Swine-pox (Variola suilla).
Camel-pox.

Though termed Variola, the identity of vaccinia and other forms in animals with the small-pox of human beings has not been clearly established. Transference of the latter to the cow is denied by many authorities, while it is contended by others that such can be effected and that vaccinia and the other forms are small-pox modified by long continued growth in different classes of animals.

The relationship is based on similar manifestations of symptoms of papules, vesicles, pustules (Pocks) and scab, and on the fact that vaccination of cow-pox lymph confers in human beings an immunity from small-pox.

During outbreaks of Variola in mules, vaccination with cow-pox lymph has been carried out and an apparent immunity from horse pox obtained.

It appears therefore that the adaptation of the virus for the various species of animals from an original type might have been possible.

Vaccination of cow-pox lymph protects against small-pox in human beings, but is useless in sheep-pox.

It is, however, certain that horse-pox and cow-pox are one and the same disease, and of a nature identical with the vaccination in human beings. They are regularly intercommunicable.

In no form of Variola has the causal agent been discovered, and until this has been accomplished, the relationship of the forms in man and animals will remain more or less conjectural. The germ is ultramicroscopic. Recent investigations incline to the belief that it is a protozoan.

Variolous and vaccine matter retains its activity a considerable time even if desiccated, but heat and the usual disinfectants readily destroy it.

For purposes of this handbook the variolas of animals most frequently met with will be considered separately.

### Horse-pox.

*(Variola equinæ.)*

**Nature.**—It is a benign, contagious disease, characterized by the formation locally, on the skin, of papules, vesicles and pustules terminating in a scab.

**Infection.**—It is only contracted by inoculation, and owes its occurrence in individual outbreaks or cases to a previous case, and possibly to a case of cow-pox or a vaccinated human being. The infective material is contained in the lymph of the vesicles or pustules. Once it is started in a stable, it readily passes from horse to horse through the medium of the hands of attendants or shoeing smiths, through clothing, brushes, sponges, rubbers, litter or anything contaminated by the virus.

The common seats are the hollow of the pastern, extending sometimes to the back of the metacarpal or metatarsal regions, and the lips and nose. In the former situation, where cracks, scratches and slight wounds are common, infection from contaminated bedding or even from handling, say in shoeing is easily understood. In the latter region, infection is either secondary from licking and biting some previously infected part, or results from a contaminated manger, nose bag, bit, etc.

**Symptoms and diagnosis.**—Febrile symptoms are either absent or very slight.

The first noticeable sign in the hollow of the pastern is heat, tenderness and swelling of the part, which in about four days begins to exude drops of a limped, slightly yellowish serosity, and which later on encrusts on the surface of the skin as a yellow mass matting the hairs together. After about ten days, under favourable circumstances, the local inflammation subsides, and healing begins. It is difficult to recognise any of the usual stages of the disease, *viz.*, papule, vesicle, or pustule, in this situation or in parts thickly covered with hair; moreover from continual stamping and rubbing consequent on the irritation which usually accompanies the disease, vesicles get broken, so that the appearance is more of a general exudation.

When the disease is located at the lips and nostrils and particularly when the buccal mucous membrane is involved, as sometimes occurs, it is possible to make out the recognized stages, first papules, then vesicles with straw coloured contents, afterwards pustules which dip in the centre, averaging about the size of pea, and lastly a scab, the whole evolution lasting from 15 to 20 days.

**Differential diagnosis.**—Since the days of Jenner it has been confused with "Grease" or seborrhœa of the digital region. It is however distinguished from the latter by its transient course, its inoculability and the abundant yellow exudate concreting on the hairs of the pastern.

It may be confused with Stomatitis pustulosa contagiosa; in fact many writers state that this disease is merely a localised form of horse-pox.

**How to deal with the disease.**

(1) Isolate affected case at once with all its belongings.

(2) Destroy by fire all bedding that has been used by the affected animal, or that has been in contact with an affected case.

(3) Thoroughly disinfect standings, mangers, line gear, clothing, grooming kit, hands and clothing of attendants, and anything, which according to the seat of the disease may have become contaminated. The virus is easily destroyed by ordinary antiseptics.

(4) Wash and disinfect the legs and faces of immediate incontacts, particularly if any abrasion exist in the heels.

(5) Treat affected parts with ordinary astringent and antiseptic dressings. External treatment only is usually indicated.

(6) Return to lines after cure, which will usually be from fifteen to twenty days.

## Cow-pox.

(*Variola Vacciniæ.*)

**Nature.**—Cow-pox is a disease affecting the udder and vulva of the cow, and is characterized by the formation of vesicles and pustules. It is often of a benign nature, but a more severe type is also encountered. It is fairly prevalent in India.

**Infection.**—Usually single cases, or at most a few cases, are met with in a dairy herd; but from neglect of precautionary measures the disease may spread widely. Infection is by inoculation, the common medium being the hands of the milker. It is thus conveyed from cow to cow, or it may be originally contracted from the vaccination lesions of human beings. Lymph from cow-pox vesicles, obtained about the fifth day from inoculated calves, forms the vaccine used for the prophylaxis of small-pox in human beings the immunity conferred being almost absolute for a period of about seven years.

**Symptoms and diagnosis.**—On the fine, hairless skin of the udder and teats, the characteristic pocks in their successive stages can be well seen. After an incubation period of about three days after inoculation (longer if the disease is contracted accidently), small pale red nodules, the size of a pea or lentil, appear on the udder and teats, particularly at the base of the latter. In one or two days these become transferred into vesicles containing at first a clear and afterwards a turbid fluid. They become depressed in the centre (umbilicated), gradually increase in size up to about the tenth day, attaining the dimensions of a kidney bean and assuming a circular shape on the udder and an oblong shape on the teats. The udder is hot and tender, and around the pocks is an area of inflammation and thickening. By the tenth day the pocks become pustules, which gradually dry up into a thick brown scab by the fourteenth day, and are easily detachable or fall off by the twentieth day, leaving a smooth pale rose, shallow depression. Vesicles on the udder may pass through these stages, but those on the teats are usually broken by the hands of the milker.

In addition lesions on the vulva, similar to those on the udder are often present. The pustules may extend for some distance into the vagina, giving rise to a purulent discharge.

In the benign type there is little or no constitutional disturbance and fever is not always present. In the more virulent type there is considerable constitutional disturbance and temperatures may rise to ·106° with the usual attendant signs such as dullness, disinclination to feed and reduction in milk yield. In the most acute outbreaks deaths have been known to occur in animals in which no complicating protozoal infection could be determined, and were apparently a direct result of the acute form of the disease.

Considerable swelling of the teats may occur and the lesions may form abscesses involving large areas of the mammary glands and extending along the subcutaneous tissue of the abdomen. These cases are difficult to treat satisfactorily and often occasion considerable loss through resultant blind teats

During the course of the disease, even in mild outbreaks, there is usually considerable reduction in milk yield, the loss often being as high as 80%.

In consistency the milk is found to be watery and coagulates more easily.

## Differential diagnosis.

**Foot and mouth disease.**—The absence of foot and mouth lesions, absence of rapid spread, the umbilicated and multilocular character of the vesicle, sufficiently distinguish cow-pox from foot and mouth disease.

**Rinderpest.**—The high fever, constitutional disturbance, mortality, and the epithelial appearance of the cutaneous lesions characterize this disease.

**Varicella, chicken-pox or false cow-pox.**—This is distinguished by the unilocular character of the pock, the absence of aerola, its rapid pustulation and drying in five or six days into a thin papery crust instead of a thick firm umbilicated scab as in cow-pox. It is also liable to appear in successive crops, and thus last for several weeks.

## How to deal with a case or cases.

1. Isolate affected animals. This need not necessarily be very far away, but the milker should on no account milk healthy animals.
2. Under the personal supervision of some responsible person, thoroughly wash the hands (not forgetting finger nails) of the milker, disinfecting with a 5 per cent. solution of carbolic acid afterwards. To make sure, all milkers should be included in this washing and disinfecting process. Examine them all carefully for any signs of sores and vaccination lesions on their hands. Make them change their clothing, sending any doubtfully contaminated garments, to the wash.
3. Be most particular to collect all *jharons* or rubbers that have been used for cleaning the udders of affected cows before milking; have them well washed and run through disinfectant solution.

4. Thoroughly scald and doubly scald all milking utensils, "paying particular attention to the handles". It is not advisable to use any disinfectant that would taste or spoil the milk. Boiling water or steam is quite sufficient.

5. Wash down and disinfect the standing of the affected animal, also the drain leading from the standing.

6. Wash the udders of all healthy cows that have been milked by the milker of an affected animal. Use warm water and disinfectant not deleterious to milk, such as boric acid. The washing should be done gently, otherwise animals may refuse to give their milk.

7. **Treatment of affected.**—Remove the milk carefully, and as far as possible without touching the pocks. A tube may be used advantageously at times. In cases where the calves are allowed to suckle the mother before milking, care should be exercised to restrain the calf from undue sucking and butting the udder. The calf's lips and nose should be disinfected afterwards.

Wash the udder first with warm water and soap carefully, then apply a lotion of boric acid: dry afterwards. Boric lotion, boric powder or boric vaseline should be applied daily to the affected part. Two drams of hyposulphite of soda to a quart of water makes a good dressing. The line of treatment should be to prevent repetition and spread of the pocks on the udder so that cure may not be delayed.

No alteration need be made in the diet. Affected animals should have their own milking utensils, which should be thoroughly scalded after use.

8. After cure, say in three weeks, the affected may return to the herd. The milker's hands and clothing must be thoroughly washed and disinfected before he is allowed to rejoin the dairy.

9. **Use of milk.**—If care is exercised in milking so that pocks are not broken and the contents mixed with the milk, its consumption is not injurious, but precaution should be taken to boil it. If, however, there is no absolute necessity, it should not be used for human consumption, especially so when there is a change in consistency in the milk.

NOTE.—Before purchase of dairy cows, udders and teats should be carefully examined for any signs of vesicles or, pustules, the folds of the gland when emptied not being omitted.

## Sheep-pox.

*(Variola Ovinæ.)*

**Nature.**—It is a highly contagious, eruptive fever, peculiar to sheep, resembling small-pox in the human being, and characterized by the appearance of papules, vesicles, pustules and scabs on the fine skin of the body. It is attended with considerable mortality.

Like other variolous diseases, its permanent home is in Asia. The Madras Presidency suffered heavily in 1894-95.

**Infection.**—As in small-pox, infection is very diffusible. Absolute contact is not necessary: it can be carried in the air on dust, or otherwise. A cramped location, a dusty highway and the gregarious habits of sheep are factors in the spread of the disease. Infection is also readily carried by man, dogs, vermin, flies and insects. It has an unusual vitality, and if secluded from free air and sunshine, will remain active for a long time even if dried. Wool, skins, and manure are infective for an indefinite period, and sheep sheds may retain infectiveness for five or six months. Sheep recovered from the disease may transmit the disease for six weeks. Virulence is rapidly destroyed by free air and sunshine, a temperature of 140° F., and by the action of ordinary disinfectants.

Goats are very refractory to infection, and may even live amongst diseased sheep without contracting the disease. It is also extremely doubtful whether it can be communicated to other animals and man.

**Symptoms and diagnosis.**—Incubation is from six to twenty days, shorter in the summer than the winter.

There are two forms, *viz.*, (*a*) the discrete, regular or benign, and (*b*) the confluent, irregular or malignant. In the former the mortality is about 10 per cent., and in the latter as high as 90 per cent.

The first symptoms are dullness; the animals separate from the rest of the flock; they show little or no appetite; stiffness in gait; shivering fits due to high temperature, which runs up to 105°, 106° and 107° F; increased sensibility of the skin inside the armpits, thighs and on the lower surface of the abdomen.

In about four days small red spots or papules appear on the fine skin of the above regions, giving them a flea-bitten appearance. The eruption also affects the mucous membrane of the eyes, nose and mouth, resulting in a discharge from the eyes and nose, at first watery, afterwards purulent, and an increase of saliva.

After about three days the papules have resolved themselves into vesicles, containing a liquid, at first transparent, afterwards turbid. The characteristic sheep-pox vesicle is small (1/5 to ½ an inch) and flat, on its surface; in this particular it differs from the small-pox vesicle which is conical, and the cow-pox vesicle which is umbilcated.

In three more days the vesicle becomes pustular increasing in size, and drying up in a few more days into a greyish crust, which ultimately is detached leaving a pink pitted spot.

The duration of the disease in the discrete form is from three to four weeks. One attack confers permanent immunity.

In the confluent or malignant form fever is very high, vesicles are rarely formed, sheep lose their eyes, their wool falls off, the skin cracks in a zig-zag manner, and the nostrils become filled with a fœtid discharge. Sometimes the alimentary tract is implicated, and there is a fœtid diarrhœa. Ewes abort.

## How to deal with an outbreak.

1. Isolate affected animals.
2. Separate all animals that appear to be unwell.
3. If the disease appears to be of the malignant type, it is wise to kill the affected to save the rest of the flock and to prevent spread of the infection.
4. Remove the flock to clean pasture or locations.
5. Split the flock into small lots to prevent overcrowding especially in hot weather when the disease is apt to be more severe.
6. Warn neighbouring flock-owners to keep their sheep away and to have no communication with an affected flock.
7. Bury deeply all carcases, slashing the skin to prevent its being used.
8. Thoroughly disinfect sheds and locations, paying particular attention to ground, feeding troughs, rubbing post, fences, broken fodder or anything likely to have been contaminated. See "Routine of disinfection".

**Treatment.**—This should only be permissible in benign outbreaks. Keep the affected in a dry and cool shelter not exposed to rain or too much sun. Apply antiseptic dressings to the eruptions, eyes, nose and mouth. Keep away flies and other possible conveyors of infection.

A little nitrate of potash in the food in one dram doses is beneficial: if constipated, give 3 ounces of sulphate of soda or magnesia. Avoid giving heating agents to bring out the eruption as severity of any case and complex infections are usually in ratio to the extent of the eruption.

Give easily digested food such as bran, meal, soaked gram, green dhoob grass, linseed tea, sliced turnips, if available.

Fresh drinking water should be liberally allowed, and a little rock salt to lick.

**Prevention.**—Vaccination by cow-pox lymph is useless.

Inoculation by the virus of the disease drawn from mild cases may be practised, but it is attended with danger of spreading the disease, and should only be performed by a veterinary surgeon. It reduces mortality to two per cent. and a whole flock may be passed through the disease in about three weeks instead of the disease hanging on for three or four months. Immunity is conferred for a year or longer. The operation is performed on the lower surface of the tail near its tip, or the inside of the ear one inch from the tip (preferably the former). An ordinary suture needle or a special inoculating needle with a groove along the side, both smeared with virus, is introduced obliquely under the epidermis, and pressed on with the thumb as it is withdrawn: or the skin may be scratched as in vaccination, virus applied, and the part covered over with sticking plaster.

A serum-simultaneous method has been used with success. Barrel gave 5 to 15 ccs. of immune serum subcutaneously in the rump

and 0·5 cc. virulent lymph in the ear. Out of 10,000 sheep submitted to this method only 20 contracted the disease. Hyper immune serum alone causes an immunity lasting up to 40 days.

### Camel-pox.

(*Vernacular—Thandy, Checkah.*)

**Nature.**—It is a benign disease through which nearly all camel *bachas* pass in the first or second year of life. It is highly contagious, occurs at all seasons, but is more severe in the rains.

**Symptoms.**—In mild cases there is diffuse swelling of the lips, with the appearance in a few days of papules. Vesicles and pustules are not well marked as camels rub the affected part, but brown crusts are formed. The course of the disease is about three weeks.

In more severe cases, which are usually met with in the rains, the lesions are seen on the lips, sheath, head and feet, or may be all over the body. A certain amount of fever is present. Sometimes the conjunctiva is affected, and permanent blindness results. Seen from a distance during the crust stage, the appearance is not unlike a case of ring-worm.

### How to deal with the disease.

It is best to let it spread among the *bachas*, particularly in seasons other than the rains. Segregation need only be practised during the latter season.

Camelmen in the S. E. of Punjab and in Rajputana inoculate *bachas* in May and June when they are about five months old. The disease is then got over before the rains begin. Crusts rubbed up in milk are used, the skin of the lips is pricked, and the mixture rubbed in. Incubation is four days.

As a rule treatment is not necessary. The *dachis* should not be allowed to travel long distance otherwise the *bachas* get no rest. A little boric acid powder to the part is useful.

Adult camels should always be segregated. When the attack is severe and the animal is weak from fever a little rum in 4 oz. doses may be given.

# NOTES.



 

## APPENDIX "A"

*Monthly report on outbreak of contagious disease.*
*Month ending..................................Class of animal.*
*Disease..............................................Method of diagnosis.*

| Station. | Unit. | Remaining. | Admitted. | Total. | Cured. | Died. | Destroyed. | Remaining. |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | | |

Brief notes on history of outbreak and points of importance.

*Age and Breed of animal to be indicated in case of death.*

SUMMARY.

| Station. | Unit. | Date outbreak commenced. | Admitted. | Cured. | Died. | Destroyed. | Remaining. |
|---|---|---|---|---|---|---|---|
| | | | | | | | |

Signature of reporting officer.

No.
Date.
Copy to:—
D.V.S. in India.
D.D.V.S....................................Command/Army.
D.A.D.V.S.................................District.

## APPENDIX "B".

### Request for Laboratory Examination.

*N.B.*—This form will accompany each specimen (in duplicate) sent to the laboratory and will be signed by the V. O. or V. A. S. i/c. case. After completion of the laboratory report it will be attached to the patient's Veterinary History Sheet, where applicable.

No.

Unit.

Station and date.

1. Class of animal.
2. Unit No.
3. Caste.
4. Sex.
5. Age.
6. Unit.
7. Date of onset.
8. Date of admission.
9. Disease.
10. Nature of specimen and preservative used where applicable.

*11. Hour and date of taking specimen.

12. Examination required.

*Note.*—*In case of Anthrax or suspected Anthrax the length of time after death at which smears were taken should invariably be indicated as accurately as possible.

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Brief clinical history of the case and P. M. finding (where applicable).

Signature of V. O. or V. A. S. i/c. case.

## APPENDIX "C".

*List of materials to be collected and forwarded for Laboratory examination.*

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| 1. *Actinomycosis* . . | 1. Pus smears from lesion, and crushed granules from pus.<br>2. Pus swabs from lesion.<br>3. Piece of tissue from lesion. |
| 2. *Anthrax* . . . | *Ante-mortem.*<br>1. Unstained blood films.<br>2. Blood swabs.<br>3. Smears and swabs from serous material from throat swellings if present.<br>*Note.*—If fixation with heat of films is carried out it must be very light.<br>*Post-mortem (where carried out) from suspected cases.*<br>Several films and swabs from:—<br>(*a*) Spleen.<br>(*b*) Heart blood.<br>(*c*) Oedematous fluid, if present.<br>*Method of packing unfixed blood films.*<br>These can most conveniently be sent in a *small wooden case*, in which chloroform ampoules are issued to hospitals from the M. S. Ds. This container will hold 4 or more thin glass slides with a slip of paper between each. The receptacle can be burnt after receipt by the Laboratory. |
| 3. *Bacillary Necrosis* . | 1. Unstained smears from pus.<br>2. Swabs from lesion.<br>3. Animal Inoculation (Martial to be sent in 50% glycerine in water). |
| 4. *Blackquarter* . . | 1. Unstained smears from lesion.<br>2. Muscular tissue (1 inch square in formol saline).<br>3. Muscular tissue (1 inch square packed in salt).<br>4. Swabs from lesion. |
| 5. *Botriomycosis.* . . | 1. Smears from lesion.<br>2. Piece of tissue in 10% formol saline. |
| 6. *Bursatti* . . . | Tissues. (In formol saline.) |
| 7. *Coccidiosis* . . . | Fæces (mucous and blood especially preserved in 2·5% solution of Pot. Bichromate in water in the following way).<br>To one spoonful of fæces add 50 c.c. of the above solution. Preserve this in a well stoppered bottle and despatch immediately. |

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| 8. *Contagious abortion* .. | *Equine.*<br>1. Pipettes of serum taken under sterile precautions free from hæmolysis.<br>2. Heart blood from fœtus.<br>3. Stomach contents.<br>4. Swabs from vaginal discharge.<br>*Bovine*<br>1. Pipettes of serum taken as above.<br>*Note.*—Heart blood from fœtus and stomach contents should be sent to the Research Officer for Contagious Abortion, I. V. R. I., Mukteswar. |
| 9. *Contagious bovine Pleuro-pneumonia.* | 1. Fresh lymph from Lung Tissue—1 oz.<br>2. Piece of Lung 1 inch square in formol saline 10%. |
| 10. *Contagious Pneumonia* | 1. Small piece of Lung in 10% formol saline.<br>2. Swabs from interior of lung tissue. |
| 11. *Encephalomyelitis* . | (*Material for transmission experiments and histological examination.*)<br>(*a*) *Preserved in 50% Glycerine.*<br>(*i*) Small portion of cerebral cortex from brain.<br>(*ii*) Portions of cervical, dorsal, lumbar and sacral cord.<br>(*iii*) Medulla oblongata.<br>(*b*) *Preserved in ice.*<br>(*i*) Medulla 1/4 inch thick.<br>(*ii*) Cerebral cortex—hazel nut size.<br>(*iii*) Hippocampus.<br>(*iv*) Piece of cord from thoracic region.<br>(*v*) Cerebellum—hazel nut size.<br>Serum from acute as well as recovered cases may be sent for the purpose of serum-virus neutralisation tests.<br>These should be sent to Pathology and Bacteriology Section, I. V. R. I., Mukteswar.<br>*To be sent to M. V. Laboratory, Lahore Cantt.* |
| 12. *Epizootic lymphangitis* | 1. Pus smears.<br>2. Pus swabs from unopened lesion.<br>*Note.*—Examination of fresh smears on the spot is advisable. |
| 13. *Foot and Mouth Disease.* | Blood smears as soon as a secondary rise of temperature or onset of complicating symptoms is noticed.<br>If alimentary tract is involved, in such complications fæcal examination should be carried out on the spot and action taken to despatch specimens of fæces (see Coccidiosis). |

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| 14. *Glanders* . . . | 1. *Ante-mortem.*<br>Swabs and smears from unopened farcy lesion.<br>2. *Post-mortem.*<br>Small piece of lung in formol saline.<br>Small piece of lung in 50% glycerine.<br>Small piece of nasal septum. |
| 15. *Hæmorrhagic Septicæmia.* | *Ante-mortem.*<br>Unstained blood films.<br>Blood swabs.<br>Films and swabs from œdematous swellings when present.<br>*Post-mortem.*<br>Films from heart blood.<br>Swabs from œdematous fluid from swellings or heart blood. |
| 16. *Jhooling* . . . | Portions of affected skin and subcutis. |
| 17. *Johne's Disease* . . | 1. Smears from rectal mucous membrane, and from pinched off pieces of mucous membrane.<br>2. Fæces.<br>*Post-mortem.*<br>Piece of bowel (ileum) and mesenteric gland in 10 per cent. formol saline. |
| 18. *Piroplasmosis* . . | G. T. test on the spot in case of equines. Examination of blood smears on the spot and despatch to laboratory where necessary. When smears are sent to Laboratory, results of examination of smears carried out on the spot should be recorded in the *pro forma*. Thin smears should be taken on clean glass slides. |
| 19. *Calf Pyosepticæmia* | Blood films.<br>Serum in pipettes.<br>Blood cultures.<br>*Post-mortem.*<br>Portions of:—<br>1. Lung containing abscesses.<br>2. Abomasum and intestines showing alteration.<br>3. Spleen, kidney and Liver, preserved in 5% formalin or formalin saline. |
| 20. *Rabies* . . . | Whole brain in case of canine.<br>Half brain in case of herbivora. |
| *Note.*—When human beings are involved specimens should be sent to the nearest local medical lab. (Bde. or Dist.). | Method of packing and preservation is given in the chapter on Rabies. |

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| 21. *Rinderpest* . . . | *P. M. material only.* Portions of affected abomasum and bowel in formol saline. |
| 22. *Tetanus* . . . | Smears and deep swabs from wound if present. |
| 23. *Thieleriasis* . . . | Thin blood films. Prescapular gland smears. P. M.:—Portions of—Abomasum, Lymph gland and Kidney. |
| 24. *Trypanosomiasis* . . | Citrated blood if in vicinity of M. V. L. Thick blood films, and ordinary films. *Camels*:—Serum for M. C. test if not done locally. |
| 25. *Tuberculosis* . . . | Sputum and milk samples. *P. M.* Pieces of lung, liver, spleen, and lymph gland showing lesions in 10% formol saline. Pieces of diseased or suspected tissue in 50% glycerine in water for animal inoculation test. |
| 26. *Ulcerative lymphangitis.* | Smears and swabs from unopened lesions. |
| 27. *Variola*:— Cow pox . . . . Horse pox . . . Sheep pox . . . Goat pox . . . Swine pox . . . Camel pox . . . | Pock material in 50% glycerine, when required. |
| 28. *Ticks* (Method of removal). | Grasp it between the thumb and forefinger, with the nail of the latter just over the capitulum and then exert gentle pressure on the body of the tick. This will prevent the mouth parts (Cheliceræ and hypostome) from being torn off, as not frequently happens when force is resorted to. *Preservative*:— Preserve in 70% alcohol, some crumpled tissue paper being placed in the tube to prevent the specimens from rocking. |
| 29. *Insects* . . | Pinned to the inner surface of the cork in an ordinary specimen tube. *Winged specimens*:—Whilst despatching by post the pinned specimens should be packed in a suitable packing case with a large quantity of resilient material for being dry and brittle as they are, are very liable to be damaged by rocking in transit. |

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| | *Without wings*:—Lice and fleas and the larval forms of insects should be sent preserved in 75% alcohol, some crumpled paper being used to pack them down. |
| 30. *Scrapings for identification of acari.* | Scrapings should invariably be made as deep as possible and may be preserved in 75% alcohol.<br>A label giving particulars (written in pencil) in regard to host, locality, date and the collector's name should be placed in the tube, in addition to the usual *pro formas* sent to the Laboratory. |
| 31. *Stomach and Intestinal contents for suspected poisoning.* | Rectified or methylated spirit may be used as preservatives when sending specimens suspected to contain non-volatile poisons.<br>In case of volatile poisons a saline solution made up of pure water and ordinary salt in the proportion of one teaspoonful to 1 pint should be used.<br>Formalin should *not* be used.<br>A list of volatile poisons likely to be met with in India is as follows:—<br>1. Prussic acid and its salts.<br>2. Alcohol.<br>3. Phenols.<br>4. Sulphuretted hydrogen.<br>5. Phosphorus.<br>6. Nicotine.<br>7. Conine.<br>8. Arecoline.<br>9. Turpentines. |

MGIPC—L—I-4-3(PDArmy)—15-2-45—1,500.